ILLINOIS MEDICAL JOURNAL

THE OFFICIAL ORGAN OF

THE ILLINOIS STATE MEDICAL SOCIETY

VOL. LXIV | OAK PARK, ILL., December, 1933 | No. 6

ILLINOIS MEDICAL JOURNAL

Published monthly by the Illinois State Medical Society under the direction of the Publication Committee of the Council.

GENERAL OFFICERS, 1933-1934

PRESIDENT.....PHILIP H. KREUSCHER, Chicago
PRESIDENT-ELECT.....CHARLES D. CENTER, Quincy
1ST VICE-PRESIDENT.....C. G. FARNUM, Peoria
2ND VICE-PRESIDENT.....H. V. GOULD, Chicago
SECRETARY.....HAROLD M. CAMP, Monmouth
TREASURER.....A. J. MARKLEY, Belvidere

THE COUNCIL

E. H. Weld,	1st District, Rockford	1935
E. C. Cook,	2nd District, Mendota	1935
R. K. Packard,	3rd District, Chicago	1935
J. S. Nagel,	3rd District, Chicago	1934
L. E. Day,	3rd District, Chicago	1936
E. P Coleman,	4th District, Canton	1934
S. E. Munson,	5th District, Springfield	1934
T. B. Knox,	6th District, Quincy	1936
I. H. Neece,	7th District, Decatur	1934
C. E. Wilkinson,	8th District, Danville	1935
Andy Hall,	9th District, Mt. Vernon	1936
J. S. Templeton,	10th District, Pinckneyville	1936
Edw. S. Hamilton,	11th District, Kankakee	1935

Chairman of Council, R. K. Packard

EDITOR

CHARLES J. WHALEN.....25 E. Washington St., Chicago

GENERAL COUNSEL

EDWIN W. RAWLINS.....105 S. La Salle St., Chicago

PERMANENT HISTORIAN

IRVING S. CUTTER.....301 East Chicago Ave., Chicago

MEDICO-LEGAL COMMITTEE

J. R. BALLINGER, *Chairman*.....2724 W. North Ave., Chicago
R. O. HAWTHORNE, *Secretary*.....Kankakee

EDUCATION COMMITTEE

MISS JEAN MCARTHUR, *Secretary*.185 N. Wabash Ave., Chicago

SCIENTIFIC SERVICE COMMITTEE

FRANK L. BROWN, *Chairman*....4034 W. Madison St., Chicago
HAROLD M. CAMP, *Secretary*.....Monmouth

PUBLICATION COMMITTEE

HARRY J. STEWART, *Secretary*.....715 Lake St., Oak Park

Outside of editorial or allied views or statements that are the authoritative actions of the Illinois State Medical Society, the organization denies responsibility for opinions and statements published in the ILLINOIS MEDICAL JOURNAL. Views expressed by the various authors and views set forth in various departments in the JOURNAL represent the views of the writers.

State Society will pay no bills for legal services except those contracted by the Committee. Notify the Chairman at once. Do not employ attorneys.

Send original article, advertising copy, cuts and all communications relating to advertising to Dr. Charles J. Whalen, c/o ILLINOIS MEDICAL JOURNAL, 185 N. Wabash Ave., Chicago.

Membership correspondence to Dr. Harold M. Camp, Monmouth, Ill.

Society proceedings and news items and changes in the mailing list to Dr. Henry G. Ohls, Managing Editor, 1618 Juneway Terrace, Chicago.

Contributors will submit all copy for publication typewritten on standard size paper and double spaced. Copy not complying with this rule will be returned, if convenient.

Subscription price of this JOURNAL to persons not members of the Illinois State Medical Society is $3.00 per year, in advance, postage prepaid, for the United States, Cuba, Porto Rico, Philippine Islands, Hawaiian Islands and Mexico. $3.50 per year for all foreign countries included in the postal union. Canada, $3.25. Single current copies, 50 cents.

Editorials

CANCER CONTINUES AS A PERPLEXING PROBLEM, BUT HAS CEASED TO BE AN ALTOGETHER BAFFLING PUZZLE

CANCER THE CURSE CAN BE LIFTED

Cancer continues as a perplexing problem but has ceased to be an altogether baffling puzzle.

Early diagnosis and immediate and drastic treatment appear to point the way out. This in the face of statistics that show cancer to have increased during the past ten years at a rate twice as fast as the sister scourge, tuberculosis, has declined. Three decades ago the tuberculosis situation was much as the cancer situation is today, with the scales in favor of the current cancer situation.

Consensus of opinion today is that cancer is curable if: 1. Discovered in time; 2. Diagnosed accurately; 3. Treated immediately and faithfully.

This too in the face of the fact that mortality from cancer has both increased rapidly during recent years and is still on the increase and as yet presents no definite isolation of germ, source or contributory cause. Cancer, like the mind, comes at its own behest so far as accurate mortal knowledge extends. Yet cancer statistics are nothing if not illuminating with the lamp of hope shedding the light on the future. Delayed treatment seems to be the greatest obstacle. What figures are available on the cancer situation are crowded with interest.

For example: With respect to curability there is little room for doubt provided appropriate treatment is undertaken early enough in the course of the disease. Definite and detailed records, show 8,840 people cured of cancer, many of whom are still alive and well. Last year, at the annual clinical congress of the American College of Surgeons in Boston, eminent surgeons from all parts of the United States gave detailed reports on 4,348 cases of cancer which have been

cured. Each case has remained well and healthy for five years or more after the treatment. Diagnosis of cancer in each case was verified by all available methods and by the opinion of specialists.

Further, on file with the secretary of the American College of Surgeons, are records of another 1,263 people who have been cured of cancer and who were alive and healthy five years or more after treatment. Each case was authentic cancer, verified by the opinion of specialists and by laboratory examination.

A third group of 3,089 people recovered completely of cancer is made up of records collected from various medical journals and books. Each was undoubtedly cancer. Each person remained well and healthy for five years or more after treatment for cancer. A fourth group of 140 cases reported by individual physicians who handled the patients and who supplied satisfactory proof that cancer was the trouble in each case states that the persons recovered and remained well and healthy for at least five years after treatment. Aggregate of these four groups brings together records of 8,840 people who undoubtedly had cancer and who recovered completely after appropriate treatment. Surgery, radium and x-ray were the methods used in the treatment of these patients. These are the only methods known to be effective in the cure of cancer.

Mortality from cancer is on the increase. In Illinois, for example, the death rate has gone up from 82 per 100,000 population in 1918 to 114 in 1932. From about 5,000 per year a decade ago, the number of deaths from cancer has climbed to about 9,000 now in Illinois. Increase is more rapid among men than women. While nearly all cancer mortality is among people over 40 years old, the death rate has increased much more rapidly than the proportion of people in the cancer age, and much more rapidly than the improvement in diagnostic methods.

The conclusion is, that cancer death rate has increased because appropriate treatment has not been resorted to early enough. Why cancer prevalence is on the increase nobody knows.

Cancer is not confined to humans. All forms of life, both animal and vegetable, are subject to cancer. There is no evidence that cancer is on the increase among lower forms of animal life. Indeed the contrary is true. Autopsies performed on more than 5,000 birds and mammals that have died in the Philadelphia zoological gardens, show that cancer is the cause of death in 2 out of each 100. Also that no significant increase in the prevalance of cancer has taken place among birds or mamals during the last decade.

Among humans, on the other hand, about 12 out of each 100 people who die are the victims of cancer.

Dublin has shown that mortality from cancer is much greater among wage earners than among business and professional groups. For wage earners the death rate from cancer is 25 per cent higher than it is among professional and business people, the white collar class. Dublin's statistics show that cancer of the mouth is twice as frequent among wage earners as among business and professional groups.

In this connection it is noted that other studies show uncorrected dental defects are much more frequent among wage earners than among those with greater incomes and no less than 9 per cent. of recruits for the navy were rejected because of dental defects. Appropriate treatment of cancerous conditions in places like the mouth where detection is facile early in the progress of the disease, will result in complete recovery in the great majority of cases. Dental defects and other causes or irritation set up cancerous conditions which do not receive prompt attention and therefore lead ultimately to a fatal outcome.

Illinois alone has some 27,000 cases of cancer with a majority age content of over forty. Of this number about 20,000 cases are in the early stages and about one-half of the women and about one-third of the men in this group of 27,000 have the disease in accessible parts of the body, mouth, breast, skin, rectum, cervix. The early symptoms can therefore be detected.

Now the average person with cancer waits for five and one-half months after he notices the first symptoms before he consults a physician about the trouble. Usually another delay between the date of first consulting a physician and the beginning of appropriate treatment, results only too often in the fatal progress of can-

cer. At the turn of the century it was known to the medical profession that tuberculosis is caused by the tubercle bacilli but how to use that knowledge for the benefit of patients was debatable.

The public believed then that tuberculosis was hereditary and uniformly fatal. The death rate was appalling, much higher than that from cancer today, at least in Illinois.

Today diagnosis is established in tuberculous patients at dates earlier in the course of the disease than was *dreamed* possible thirty years ago. Courageous physicians took the knowledge at hand thirty years ago and attacked the problem of tuberculosis with eloquently justified results. Prevalence of the disease is reduced to a point far beyond the hope and the belief of even the most ardent a quarter of a century ago. Attitude of the public in respect to this disease has been entirely changed.

What has been done with tuberculosis can be done with cancer. At least in so far as early treatment following diagnosis is concerned. Vigilance must be unremitting.

Not is it amiss to cite the celebrated "cancer comparison" of two ex-presidents of the United States—Gen. Grant and Grover Cleveland. Grant recognized trouble in his throat. His physician suspected cancer and advised action on that basis. For one reason or another about four months elapsed between the date when his physician advised consultation with a specialist and the time when an operation was done. Within eight months Grant was dead.

Cleveland recognized an abnormal condition in his mouth that failed to clear up within a reasonable time. His physsician suspected cancer. The diagnosis was confirmed. The President acted at once upon the advice of specialists. Complete recovery and a healthy life of fifteen years followed the operation. The operation made necessary an artificial jaw but it added to Cleveland's life a period equal to one-fourth of the average life span.

THE PRESENT DAY TENDENCY OF PASSING THE QUESTION OF DIAGNOSIS UP TO THE LABORATORIES

Modern trend of disclaiming responsibility finds annoying demonstration in the tendency of passing the question of diagnosis up to the laboratories and to a retinue of specialists where only too often the same results could be obtained from a good clinical diagnosis effected by the skilled general practitioner.

For a great many years general practitioners pursued that course. The mortality statistics were no higher, excluding sanitary control and immunization, then than they are now, or at any rate not markedly so, if at all, in comparison with the red tape and the general expense to which the patient is put. Because our young men are not taught to make clinical diagnosis, as a consequence the patient must pay for laboratory work without end. The same condition appertains to the matter of treatment. Special treatments are in the same status with all these laboratories where expense to the patient is concerned and without any appreciable advantage save in semi-rare instances. Instead of considering the body as a whole, the patient is told he must have a specialist for every section of his anatomy and for cross sections of that. All in all a wholly absurd procedure for the average ills of mankind.

When the general practitioner made his fairly good diagnosis he was wont to obtain like results in the majority of instances. Today we are taught and told that no one man can make even a working diagnosis and that our patient *must* receive attention of numerous specialists. Doctors of years gone by seemed able to meet all the therapeutics that might arise. Experience and observation says this was done with success. Turning the patient over to this or that specialist for any one part of the patient's anatomy involved, for special treatment, has added to the charges to the patient and has not really pleased him. Neither has it in the long run decreased our mortality tables. People are still dying in about the same ratio to population as has been the case for the past forty or fifty years. There are exceptions in connection with specific diseases for which have developed specific treatment. In diphtheria has come a decrease in deaths due to the use of antitoxin. Several other diseases offer similar experiences. These exceptions have been offset in the case of other conditions in which are decided increases in the number of deaths. Without showing much final im-

provement, all this additional work has forced up medical charges to the extent that dissatisfaction is appearing in the minds of the public. There is today too much "fussing" in practically all instances and that is too expensive to the patient.

SIGN AND RETURN THE A. M. A. DIRECTORY CARD

The thirteenth edition of the A. M. A. Medical Directory is scheduled to appear about July, 1934. The last edition was issued in 1931. Recently each physician in Illinois was furnished with a card upon which to record data pertaining to his professional status. There is a crying need for a new up-to-date directory; it is essential that data included in the directory be correct.

We are informed that many physicians received the cards sent by the A. M. A. but have not filled out and returned them. We urge every ethical physician in Illinois to promptly fill out and return the data card to the American Medical Association, 535 North Dearborn Street, Chicago, Illinois.

If any medical reader failed to receive such card, he should write to the American Medical Association, 535 North Dearborn Street, Chicago, Illinois, calling attention to the fact.

There was never a time when it was more important for every active physician to be a member of his County and State medical organizations and a Fellow of the American Medical Association.

STRESS FOR NEED OF FREQUENT MEDICAL EXAMINATION

Indubitably more stress should be placed upon vital need for more frequent physical examination.

Findings of accident actuaries employed by the large casualty companies indicate that there is an alarming increase in accidents occurring in the home and that also during 1932 the rate from fatal accidents *in the home* was the highest on record. Further during the years running between 1924 and 1932 the rate from fatal accidents in the home among persons at ages fifteen or over increased something well over 41 per cent. There was no such increase but rather a definite decrease in the home casualty rate as applied to children. Whether the acceleration of this hazard lies in the fact that the economic situation and loss of employment everywhere has kept more adults in the home or whether it is a reflection of an adult physical condition is to be considered. Since the increased hazard appears to be due entirely to accidents involving falls, and to falls among adults the finger would seem to point to some contributory physical defect or diversion incidental to maturity. In the year 1924, for example, when this increase began to be shown one-third of all fatal accidents occurring to adults while engaged in activities in and about the home were due to falls while in 1932 this single class accounted to *fifty* per cent. of all domestic accidents. Now with respect to accidental burns, the death rate has remained on practically the same level throughout the nine-year period, while asphyxiations by gas are fewer than ever before. In the miscellaneous class of casualties the group rate varies but triflingly either above or below the accepted average. In this class "miscellaneous" are considered casualties other than falls, burns and asphyxia by gas.

Any number of serious illnesses may evidence their presence by that loss of equilibrium and muscular co-ordination that leads to a fall. Especially a fall in such familiar terrain as the home even allowing a certain discount for natural human carelessness. The way to exercise prevention is by investigation—before and not after the accident.

In this instance the barn door is best locked by frequent and thorough physical examination

FATAL ACCIDENTS IN THE HOME

Kind of Accident; Age	Deathrate from Home Accidents per 100,000 Persons								
	1932	1931	1930	1929	1928	1927	1926	1925	1924
All Home Accidents									
Under 15 years	11.1	11.2	11.2	13.5	13.3	14.2	16.3	16.5	14.4
15 years and over	15.6	15.2	14.4	12.7	11.7	12.3	13.0	12.6	11.0
Burns (conflagration excepted)									
Under 15 years	4.6	4.5	5.3	6.1	6.0	6.4	7.4	7.4	6.3
15 years and over	2.1	2.0	2.1	2.2	2.1	2.0	2.2	2.3	2.2
Traumatism by fall									
Under 15 years	1.8	1.8	1.4	1.7	1.5	1.9	2.0	2.0	1.8
15 years and over	8.8	8.1	6.9	5.6	5.0	4.9	4.6	4.4	3.8
Absorption of poisonous gas									
Under 15 years	.3	.3	.2	.5	.3	.2	.6	.4	.5
15 years and over	1.8	2.2	2.4	2.6	2.8	2.8	3.7	2.7	3.0
All other accidents									
Under 15 years	4.4	4.6	4.3	5.2	5.5	5.7	6.3	6.7	5.8
15 years and over	2.9	2.9	3.0	2.3	1.8	2.6	2.5	3.2	2.0

of adults. The conscientious family physician will insist upon this.

The appended table has been issued by the Metropolitan Life Insurance Company:

DOCTORS DESIRING TO PRESENT PAPERS AT 1934 ANNUAL MEETING

Members of the Illinois State Medical Society desiring to present papers at the 1934 Annual Meeting to be held in Springfield, May 15, 16, 17, 1934, should get in touch with the proper section officers as soon as possible.

Owing to the large membership of the Society, it is desirable to place members on the program each year who have not appeared in recent years.

The number of speakers at the annual meeting for each section is limited; consequently it is desired that all papers should be of interest to the members in general. Any member desiring to make a presentation at the annual meeting in any of the five scientific sections should write the Section Officers, giving the title of the paper, and an abstract of the same. If interesting case reports are desired to be given, tell briefly about the case of unusual interest, in making application for the place on the program.

It is the desire of all officers of the Sections to make the 1934 Annual Meeting program an outstanding one, and arrange it to the best advantage of all members. On Thursday morning, the last day of the meeting, it is planned to have a joint session of all five scientific sections, and present papers which will be of interest to all members regardless of their own special inclinations in practice.

Members desiring to make a presentation of either a paper or interesting case report, should get in touch with the proper Section Officers at an early date. The officers of each Section are herewith given.

Section on Medicine

R. F. Herndon, Chairman, Springfield.

Don C. Sutton, Secretary, 30 North Michigan Blvd., Chicago.

Section on Surgery

George W. Post, Chairman, 4010 West Madison Street, Chicago.

B. V. McClanahan, Secretary, Galesburg.

Section on Eye, Ear, Nose and Throat

George S. Duntley, Chairman, Macomb.

O. B. Nugent, Secretary, 231 West Washington Street, Chicago.

Section on Public Health and Hygiene

J. Howard Beard, Chairman, Urbana.

Lloyd Arnold, Secretary, 1817 West Polk Street, Chicago.

Section on Radiology

Robert F. Arens, Chairman, 2839 Ellis Avenue, Chicago.

F. Flynn, Secretary, Decatur.

LECTURES ON CATARACT EXTRACTION

Professor A. Elschnig of Prague will give a course on intracapsular extraction of cataract on Tuesday, February 27, 1934, in Chicago.

The number of registrants will be limited and there will be a registration fee of $10.00. There will be a lecture early in the afternoon, illustrated with slides and movies, which will be followed by operations on patients before groups of not more than 10 registrants. Monday evening, February 26, 1934, Professor Elschnig will speak on "Myopia Extraction" before the Chicago Ophthalmologic Society.

Send checks or communicate with Dr. Theodore M. Shapira, 58 East Washington Street, Chicago, State 5893.

HAMMER TOE WITH ANKYLOSIS

In cases of hammer toe with ankylosis it is desirable to lay the joint open and excise the ends of the bones at the joint—the base of the medial phalanx and the head of the proximal phalanx. No attention is paid to the periosteum. A simple resection clear across the two bones is made, the cut ends are placed in apposition and allowed to grow together. This results in a stiff joint it is true, but the toe is straight and the fact that it is stiff causes no inconvenience whatever.—Dr. E. W. Cordingley, of Clinton, Ind., in *Med. Herald,* May, 1932.

HEMOPHILIA TREATED BY VENESECTION

A case of long-standing hemophilia was relieved by bloodletting. Withdrawals of 500, 250 and 300 cc. of blood were made in hospital and, after discharge, of 500 to 600 cc. every five or six weeks. Headaches, vertigo, joint pains and subcutaneous hemorrhages ceased.—Dr. G. B. Lawson and associates, of Roanoke, Va., in *J. A. M. A.,* Apr. 23, 1932.

In the same Journal and issue, Drs. A. G. Foord and B. R. Dysart, of Pasadena, Cal., report a case of clinical hemophilia, actively bleeding following tonsillectomy, treated by one injection of ovarian extract, with prompt arrest of the bleeding.

Correspondence

HOME CARE

Attracted by a sign "Medical Center," I stepped into a suite occupied by two general practitioners, one holding office hours in the morning and the other in the afternoon, and a dentist who was there all day. All three worked together in the evening. This little group serving an industrial neighborhood are caring for all minor and even moderately severe cases at the office or at the patient's home. And they are doing good work.

Following a period when specialism was rampant, elaborate laboratory equipment considered essential, and hospital residence required, seemingly for every ailment, it is refreshing to find rapidly increasing emphasis placed on the value of office and home care and the quality of service that may be thus rendered to the sick person by the doctor practicing general medicine. Why should hospital expense be added to the cost of caring for a thrombotic hemorrhoid, a sebaceous cyst or a chalazion. From many sources comes the information that from 80 to 85 per cent. of all ailments can be treated at home or in the doctor's office with no more equipment than a physician usually has or may have in his office or in his hand bag.

These statements are in no way derogatory to the quality of hospital service when it is rightly needed, nor to the contributions of scientific progress made by these institutions through their special staffs. They add force to the doctor's efforts to secure comfort and cure for patients in the least time and with no unnecessary expense. They emphasize the views of those doctors who believe that the development of medical science is not for its own inherent pleasure but for the good of the people and that no sick human being may ever be regarded as an "interesting specimen."

Home nursing and social service organizations have revealed how much can be done for patients outside of institutions, and with much saving to the family and also to the community. The financial costs are not the only problem; there is the recognition of the value of personal solicitude and love in the home treatment and care of the sick. Since the personal relationship of the doctor and his patient is so valuable, much may be contributed to a patient's speedy recovery by faithful service of those vitally interested in his cure. Nor may one forget the salutary influences that react on a household and family where sickness is present and lovingly attended.

PUBLIC POLICY COMMITTEE,
Illinois State Medical Society.

BLANKS FOR SCIENTIFIC EXHIBIT AT A. M. A. 1934 CONVENTION

Application blanks are now available for space in the Scientific Exhibit at the Cleveland Session of the American Medical Association, June 11 to 15, 1934. The Committee on Scientific Exhibit requires that all applicants fill out the regular application form and requests that this be done as early as convenient.

The final date for filing applications is February 26, 1934. Any persons desiring to receive an application blank should address a request to the Director, Scientific Exhibit, American Medical Association, 535 North Dearborn Street, Chicago, Illinois.

Medical Economics

MEDICAL CARE OF THE INDIGENT

The problem of the care of the indigent has been one confronting the medical profession from time immemorial, and during the past three years, has been a bigger problem than ever before.

Physicians unconsciously classify their patient in three classes:

1st. Those who pay their bills in full.

2nd. Those who pay part of their bill, through inability to pay all of it.

3rd. Those who pay nothing at all for their medical care.

In the third class, we find those people who are temporarily out of employment, and are absolutely unable to pay for their medical care at this time, although when working do the best that they can. Again we have in this class those people who would not pay anything that can be avoided, and these are the ones who have long been considered as "dead-beats."

We all realize that in the giving of medical care, there is always a double obligation, that is, one on the part of the physician, who agrees to give good, conscientious and adequate service to his patients, and on the part of the pa-

tient to pay for these services to the best of his ability.

In Illinois the obligation of caring for their poor rests on the shoulders of the Township government, so long as they are able to pay the bills. For years, it has been the practice of many township supervisors to ask physicians to submit contracts for the care of the poor, medically, and it is almost invariably the practice to give the contract to the lowest bidder with little if any consideration to the ability of the individual to give adequate medical service.

No one can anticipate the medical needs of a community at any time, and with the large number of unemployed, and with many existing deficiencies in diet among these people, it is quite obvious that many more deficiency diseases will be found than other normal conditions, which means a greater susceptibility to disease, especially among children. The Government has recently placed medical care on the list of necessities, along with food, clothing and shelter, which must be provided for unemployed indigents.

The supervisor who believes that medical care should be provided on a contract basis, does not ask the grocer, coal dealer, and clothier to submit a contract guaranteeing the provision of these necessities to the unemployed, but they are paid for on an individual basis in every instance. A recent survey made throughout Illinois in an effort to get reliable data concerning the giving of medical care to the indigent, shows that in many counties all physicians refuse to submit a bid for a contract to care for the indigent, and in these counties subsequently, all physicians are permitted to give the necessary care upon authorization by the supervisors, and each of them receives some remuneration.

Some counties report that 100 per cent of the regular rate is allowed, while in others, a reduction is made from normal rates for service.

In checking over these reports, it is evident that physicians in all communities where no contracts exist, are receiving a better percentage of remuneration for indigent care, than where contracts are in force.

Although at this time, the reports received are not sufficient to give complete information on this phase of medical care, the Committee on Medical Economics is receiving data which will be of much value to our economics contribution within a short time. We again urge all county society secretaries to give the desired information as soon as possible, for the benefit of all physicians in Illinois.

As previously reported, during the month of September, the Federal Relief Administration released Rules and Regulations No. Seven, governing the giving of medical care to recipients of unemployed indigents, and in this, each State Medical Society was asked to submit their program to the State Emergency Relief Commission, for their approval. After approval is given to a State Society Program, it should then be approved by all County Medical Societies, and then be submitted by them to local relief agencies.

The State Society was asked to select a committee to confer with State Relief Officials on matters of medical policy, and the County Societies similarly were asked to name a committee to confer with local relief officials. The Program of the Illinois State Medical Society has been submitted to the proper officials, and full reports have been sent to all component societies. Owing to other pressing duties, the Relief Commission has not yet given their approval to this program, but we have been given every assurance that it will be attended to within a short time.

It is generally believed that when this program is approved and placed in operation, it will enable physicians in every county in Illinois to receive better remuneration for medical care of the indigents, and will aid county societies and their committees to confer with the proper officials in their community, so that unfair contracts for furnishing indigent care will be abolished.

The Committee on Medical Economics of the Illinois State Medical Society is interested in the problems of the Medical Profession, and is always anxious to do everything possible to aid in the betterment of conditions pertaining to medical practice. This committee will appreciate any suggestions from any county medical society or individual members at any time. We again want to urge the Secretaries to send to the Secretary of the Illinois State Medical Society all possible data concerning the medical care of the indigent, the plan in operation in your re-

spective communities for giving this care, and information relative to contracts given to individual physicians, all of which is important in our present medical economics survey of the entire state.

E. S. HAMILTON, Chairman,
Kankakee, Illinois.

COURSES IN OPHTHALMOLOGY

An unusually comprehensive didactic course of instruction in Ophthalmology is to be offered during the year 1934 starting January 1 to internes and residents in Ophthalmology in the various hospitals in Chicago. It is an outgrowth of and amplification of the course previously known as the city-wide course.

Two hundred hours of didactic instruction will be given by fifty-three instructors from the various universities and hospitals. These lectures will be divided evenly between the fundamental and the clinical branches, but they will in no wise be a substitute for actual clinical work.

The schedule is as follows:

Anatomy-Gross, Microscopic, Embryology, Central Visual Pathways	16 hrs.
Physiology	12 hrs.
Physiological Optics	12 hrs.
Perimetry	6 hrs.
Refraction, including spectacle fitting	12 hrs.
Muscles	10 hrs.
Pathology	14 hrs.
Bacteriology	2 hrs.
Pharmacology	2 hrs.
Principles of the Slit Lamp	4 hrs.
Instruments of Precision	2 hrs.
Methods of Clinical Examination	4 hrs.
Keeping of Records	2 hrs.
Use of the Library	2 hrs.
Diseases of the Eye	48 hrs.
Relation to Other Branches of Medicine	20 hrs.
Therapeutics—Medical	8 hrs.
Surgical	16 hrs.
Industrial Ophthalmology	2 hrs.
Prevention of Blindness	2 hrs.
Ophthalmological Ethics	2 hrs.
Fitting of Prostheses	2 hrs.
	200 hrs.

All ophthalmological internes and residents and bona fide assistants in ophthalmological clinics are invited to attend. Owing to limited space no others can be accommodated.

Practically all of the lectures will be given at the Illinois Eye and Ear Infirmary. A noteworthy departure and special feature will be the availability to the students *before the lecture* of the notes on that lecture. This will allow the student to give his undivided attention to the lecturer.

RESOLUTIONS ON THE DEATH OF DOCTOR NELSON

WHEREAS: The inevitable Fate that resistlessly awaits all men at the nether end of life has taken recently from our midst a warm friend, an able colleague, and a wise counselor in the person of Dr. Charles S. Nelson; and

WHEREAS: Throughout his long and useful professional life Dr. Nelson was tireless in his efforts to promote public faith and confidence in the ethical medical profession and as a life-long member of this Society was ever alert in the interests of the practicing physicians as represented by the Illinois State Medical Society and all of its component parts; and

WHEREAS: Dr. Nelson exemplified through his thirty odd years of service on the professional staff of the State Department of Public Health those qualities of efficiency, integrity, honesty competency and diplomacy that go to make up a physician of parts and cause to be reposed in him the respect of his fellow practitioners and the confidence of the public; now therefore, be it

RESOLVED: That the Sangamon County Medical Society expresses, herewith, its appreciation of the value and worth of the life of Dr. Nelson both as a friend and as a doctor and be it further

RESOLVED: That these resolutions be spread upon the minutes of the Sangamon County Medical Society and that a copy thereof be forwarded to the Illinois State Medical Society with a request that they be published in the ILLINOIS MEDICAL JOURNAL.

Homer MacNamara, Secretary,
Sangamon County Medical Society.

VACCINE PREVENTION OF WHOOPING COUGH*

In 1928 I began a study on immunizing 294 selected young children against whooping cough. Their ages ranged from nine months to several years and their past medical histories were familiar to me from their infancy. The vaccine was especially prepared by my collaborator from five to seven strongly hemolytic strains of the Bordet-Gengou bacillus.

The children were treated in three groups and each child was given between 7 and 8 cc. of vaccine (70 to 80 billion bacilli). The 109 children in Group A were given about 1 cc. hypodermically in alternate arms each week, until eight injections were given. The 97 children in Group B were given 1 cc. simultaneously in each arm for four successive weeks. The 83 children in Group C were given 1, 1.5 and 1.5 cc., respectively, in each arm each week for three weeks. In neither group could the local or systemic reaction be compared in severity with the mildest smallpox vaccination reaction. White blood counts made in Groups A and B on the first and last injection days showed some increase (chiefly in small lymphocytes) in over 85 per cent of cases. The vaccine seems to influence the blood picture in the same way as does whooping cough.

General questionnaire results showed that 82 of the Group A children, 29 of the Group B children and 16 of the Group C children, were intimately exposed to whooping cough, with only one developing a questionable cough. In eight cases carefully studied, in which there was continuous exposure, one child developed symptoms similar to whooping cough, but did not have

paroxysms. Her exposure began less than two months after her injections were completed. There should be at least three months' interval to consider it a fair test of immunity.

I feel that we now have a definite means of preventing this disease, if we use vaccine prepared from recently isolated, strongly hemolytic strains of the Bordet-Gengou bacillus, grown only on human blood and the like. We can not expect results from commercial pertussis vaccines.

Louis Sauer, M. D.

Evanston, Illinois.

*J. A. M. A., Jan. 28, 1933.

STATE HEALTH OFFICERS

A special program for health officers has been arranged by the State Department of Public Health to take place in Springfield on December 14 and 15. The features of the program are symposiums on epidemic encephalitis, diet and nutrition, and school health problems. A special banquet session on the evening of December 14 with Dr. W. A. Evans and Dr. Allan J. Hruby, Chicago, and Dr. Eben J. Carey, Milwaukee, as speakers, and a luncheon at noon on December 15 followed by a round table discussion of diphtheria control are expected to be of outstanding interest to health officers. Five local health officers including Dr. H. A. Orvis, Winnetka; Dr. W. M. Talbert, Decatur; Dr. John W. H. Pollard, Evanston; Dr. E. B. Coolley, Danville; and Dr. H. O. Collins, Quincy, have been asked to participate in the round-table program.

Other guest speakers who have agreed to appear on the program include Dr. Paul J. Zentay and Dr. John Eschenbrenner, St. Louis, who are to discuss epidemic encephalitis; Dr. Arlington Ailes, LaSalle, Dr. Lloyd Arnold, Dr. Julius Hess and Prof. Lydia Roberts, Chicago, who are to speak on diet and nutrition; Dr. J. Arthur Myers, Minneapolis, Dr. Robinson Bosworth, Rockford, Dr. Thurman B. Rice, Indianapolis, and Dr. G. Koehler, Springfield, who are to speak on school health problems. Dr. J. P. Leake of the U. S. Public Health Service has been invited to participate in the symposium on epidemic encephalitis. Several members of the professional staff of the State Department of Public Health are also on the program, of which Dr. Frank J. Jirka, State health director, is general chairman.

BRANCH STATE LABORATORY

Under the immediate supervision of Professor F. W. Tanner, a branch diagnostic laboratory has been opened by the State Department of Public Health at the University of Illinois in Urbana. Specimens and communications meant for this branch laboratory should be addressed to the State Diagnostic Laboratory, Room 360, Chemistry Building, Urbana.

This newly established branch is prepared to do all diagnostic tests ordinarily performed in public health laboratories. These include the examination of blood specimens for typhoid fever, undulant fever, tularemia, syphilis and malaria; sputum for tuberculosis and pus for gonorrhea and Vincent's angina; spinal fluid for syphilis; cultures for diphtheria; animal heads for rabies; excreta for typhoid.

The Urbana branch was established in response to a demand from a large body of physicians in the central eastern part of the State. Using the laboratory at Urbana will enable many physicians to get reports of results in about one-half the time necessary when specimens are sent to Springfield from the eastern portion.

Full duty diagnostic laboratories of the State Department, in addition to the Urbana branch, are located in the Capitol Building, Springfield; Southern Illinois State Normal University, Carbondale; 1849 West Polk Street, Chicago.

RESEARCH ON GOITER

The American Association for the Study of Goiter, for the fifth time, offers Three Hundred Dollars ($300.00) as a first award, and two honorable mentions for the best essays based upon original research work on any phase of goiter presented at their annual meeting in Cleveland, Ohio, June 7, 8 and 9, 1934. It is hoped this will stimulate valuable research work, especially in regard to the basis cause of goiter.

Competing manuscripts must be in English, and submitted to the Corresponding Secretary, J. R. Yung, M. D., 670 Cherry St., Terre Haute, Ind., U. S. A., not later than April 1, 1934. Manuscripts received after this date will be held for the next year or returned at the author's request.

The First Award of the Memphis Tenn., 1933 meeting was given Anne B. Heyman, A. B., M. S., University of Michigan, Ann Arbor, Mich., "The Bacteriology of Goiter and the Production of Thyroid Hyperplasia in Rabbits on a Special Diet."

Honorable mentions were awarded J. Lerman, M. D., and W. T. Salter, M. D., Huntington Memorial Hospital, Boston, Mass., "The Calorigenic Action of Thyroid and Some of Its Active Constituents," Prof. Dr. Stefan Konsuloff, Sofia, Bulgaria, "Experimental Studies on Etiology of Goiter."

WOMAN'S AUXILIARY TO THE ILLINOIS STATE MEDICAL SOCIETY

State Board Meeting. On November 18 the Board of Directors of the Woman's Auxiliary to the Illinois State Medical Society, convened in the Stevens Hotel, with twenty-six members present. Mrs. H. I. Conn, First Vice-President, occupied the chair, the President, Mrs. Solomon Jones, being absent on account of illness. A message of regret and best wishes was wired her.

Much interest was manifested in the encouraging reports of Chairmen of Standing Committees and Councilors. The Organization Chairman, Mrs. Philip Kreuscher, reported an increased interest among county medical societies in the work of the Auxiliary and in the extension of same. Mrs. Kreuscher attended and represented the President, Mrs. Jones, at a District Convention in Centralia, November 3 and 4.

The Public Relations Chairman, Mrs. W. R. Cubbins, has contacted a variety of organizations in a

united effort to improve health conditions. The lecture by Dr. Charles F. Read in the Illinois Host House and luncheon in the Trustees' Lounge at the Century of Progress in this behalf, on October 11, were propitious events.

Mrs. H. H. Hurd, Hygeia Chairman, reported satisfactory progress in securing subscriptions and is pressing her work in every organized county.

Mrs. W. R. Rhodes, Chairman of Finance, submitted a budget outline for the year, which was accepted. The financial outlook for the organization is quite satisfactory.

The Press and Publicity Chairman, Mrs. J. P. Simonds, reports heartiest cooperation from Auxiliary workers in supplying material, and from Doctor Whalen in giving our activities publicity in the ILLINOIS MEDICAL JOURNAL.

Councilors Mrs. H. B. Henkel and Mrs. I. L. Foulon, of the Fifth and Tenth Districts, reported profitable and enthusiastic meetings with the President, Mrs. Jones attending and making an inspiring address.

Mrs. A. B. Middleton, of the Second District, has Auxiliaries in progress of organization in both LaSalle and Bureau Counties, and Mrs. E. W. Burroughs was optimistic about results of her efforts in Saline County in the near future.

A beautifully appointed luncheon was served the board members in the Boulevard Room of the Stevens Hotel, at 12:30 o'clock.

That physicians and physicians' wives are awakening to the advantage to the profession of the Auxiliaries, there can be little doubt, as all counties where these organizations exist are enthusiastic in their praise.

Respectfully submitted,

(Mrs. E. B.) Nelle F. Coolley,
Corresponding Secretary.

PROGRAM BUILDING FOR A WOMAN'S AUXILIARY

MRS. E. P. SLOAN, *Program Chairman*

A simple yet adequate guide to Auxiliary program building is to be found in our constitution which defines the organization's three-fold object as:

(a) To assist the medical profession in advancing disease prevention;

(b) To essay legislation thereto;

(c) To respond to all calls from our Medical organizations in behalf of their professional interests.

Every program given by a Woman's Auxiliary to the Illinois State Medical Society should be judged by these principles. Of any suggested line of work we should ask, "does it do any or all of the above listed things;" if the answer is "yes," then the program matter is eligible.

This pattern should be stressed because of the very real importance of the work that may be done by the Woman's Auxiliary to the Medical Association. It is wise for us to keep well within the definitely prescribed boundaries of the organization's designated task—a huge one of limitless possibilities in its own right, if seriously and scientifically attacked. It is primarily a work of education—the slow, thorough, evolutionary task of bringing to the laity a better understanding of the magnificent work of medical science.

Of utmost importance in facilitating this educational program is the need that we, the wives of Doctors, first make ourselves as intelligent as possible regarding medical matters. Research in medical literature should be the pleasurable obligation of each of us; the past history of medicine is exciting, stirring drama; its present is a colossal humanitarian order, of unsurpassable importance; its future a potential blessing to civilization that challenges our most loyal efforts in defense of its free development.

High sounding words? Perhaps. Yet literally true—and unless the wives of doctors blend this vision of the inestimable value of the medical profession with the basic factual information of health programs, we miss a great factor in any educational campaign—the driving force that springs from an adequate understanding of and a sincere belief in the work itself.

Our program should be designed to educate us in order that we may effectively educate others. For that is our chief aim—to act as the informative link between our men and the public they serve. It is a delicate work requiring an extraordinarily well informed intelligence combined with a patient tactfulness.

Despite the wide range of condition under which we live, every Doctor's wife, whether located in city or country, feels the need of counsel, of encouragement, of information regarding the best methods of making herself an effective auxiliary member. Locality must influence and alter programs. Metropolitan conditions create certain emergencies, while in rural communities other types of problems are indigenous. Naturally, each unit takes its own peculiar needs into consideration and must adjust and compromise any suggested program to meet them. However, the following listed sources suggest the fundamental ingredients upon which any county unit may work:

1. The Envelope Study Programs for county auxiliaries prepared by the National Program Committee.
2. Study of history and organization of,
   (a) State Welfare Work.
   (b) State Department of Health.
3. Use of the services of the educational committee of the Illinois State Medical Society through:
   (a) Public meetings, with health talks given by the educational committee's approved speakers.
   (b) Presentation to laity groups of educational films.
4. Miscellaneous.
   (a) Review of biographies of the great men of medicine.
   This is a beautifully exciting way to learn the romantic history of the daring science of medicine.
   (b) Constant use of Hygeia, THE ILLINOIS

MEDICAL JOURNAL, and the A.M.A. Bulletin.

(c) Participation in all approved local activities that promote better health conditions, such as Child Health Day in May; a Health Audit Survey.

In short, to be always an *auxiliary* in the literal sense of the word.

Within this skeleton framework is ample material to satisfy the varied needs and interests of any auxiliary. Not until we have exhausted its possibilities should any doctor's wife complain of a dearth of program subject matter. A studious use of these facilities, gratis to us, will go far toward promoting our growth into a real auxiliary—a helpful adjunct to the profession of which we are a proud part.

NEWS ITEMS FROM ILLINOIS AUXILIARIES

Chicago Auxiliary. Mrs. N. M. Percy, Program Chairman, reports that the Woman's Auxiliary of the Chicago Medical Society had a very successful "Laity Day" luncheon and program on Wednesday, November 1, at the Medical and Dental Arts Club. Eighty-five women were present to hear Dr. W. A. Evans, Health Editor of the *Chicago Tribune,* speak on "Health and the Depression." Presidents or representatives of Cook County Women's Clubs were guests of the Auxiliary. An announcement was made offering the services of the Speakers' Bureau of the Educational Committee of the Illinois State Medical Society. The December 6 meeting will be a Card Party and Tea at the Congress Hotel. Part of the funds raised may be used in placing "Hygeia" in schools, reading-rooms, libraries, etc., where subscriptions have been stopped for lack of funds.

Aux Plaines Branch. Mrs. H. M. Peterson, President, welcomed one hundred and seventy members and guests at the annual bridge-luncheon at the Oak Park Country Club on September 22. The regular monthly meeting was held at the Oak Park Club on October 27. A Laity Day program was presented, Doctor John Favill, Neurologist and member of the National Committee of Mental Hygiene, was the speaker. On November 24 a social meeting was held at Marshall Field & Co., with luncheon in the Wedgewood Room. The speaker was Mrs. Charles W. Leigh, former art chairman, Illinois Federation of Women's Clubs.

Jackson Park Branch. Mrs. Meyer Solomon, the President, entertained members and friends on October 18 with luncheon in her home. Dr. Henry S. Houghton, formerly Director of Peking Union Medical College, spoke on "Medical Work in Peking, China." Miss Minsen Tang, student of Social Science Administration, related some of her personal experiences as a Chinese student in America. The annual card party to raise funds for the work of the branch was held on November 15 at the home of Mrs. John J. Gill.

Englewood Branch. Mrs. D. A. Vloedman, President, reports the regular monthly meeting which was held on November 22 at the Evangelical Hospital of Chicago. Doctor R. K. Packard was the speaker. His subject was "Medical Economics."

North Shore Branch. The annual social meeting was held in April at the home of Mrs. F. O. Fredrickson, the President. Members and friends enjoyed an informal luncheon and sufficient funds were raised to meet the necessary expenses in carrying on the work of the branch. The first meeting of the autumn was held September 20 at the home of Mrs. E. W. Mueller, Past President of the State Auxiliary. Mrs. Fredrickson reports that the members of the North Shore Branch are enthusiastically working to increase their membership. This is, at present, their definite object. Mrs. Frank J. Taraba is membership chairman. Members are urged to attend and to invite their friends, who are wives of physicians, to attend all program meetings of the Central Auxiliary. At the November 1 meeting members of the North Shore branch met for a Round-Table discussion during luncheon hour.

North Side Branch. Mrs. Michael L. Mason, President, announces that the first meeting of the year was held October 9 at Henrotin Hospital, with Dr. M. L. Harris, former President of the American Medical Association, giving an adddress on "The History of Henrotin Hospital." Miss Miller, the Superintendent, showed plans for the new hospital, which will be constructed in 1934. Following a tour of the hospital, tea was served. The second meeting was held on November 13 at St. Vincent's Infant and Maternity Hospital. Sister Camilla was hostess. She presented a scenario, given by children under three years of age. After a tour of the hospital, Dr. M. L. Blatt gave a short talk on "The History of St. Vincent's." The tea hour completed this interesting program.

Douglas County. The March meeting was held at the home of Mrs. J. O. Cletcher, Tuscola. Mrs. W. C. Blaine presented a paper on immunization and showed graphs from the Illinois State Department of Public Health which indicated the incidence of smallpox in Illinois compared with other states having compulsory vaccination laws. She had interesting charts showing the incidence of communicable disease by age groups and the incidence of diphtheria in orphanages before and after the administration of toxin-antitoxin and toxoid. The April meeting was held at the home of Mrs. W. C. Blaine, Tuscola. Mrs. C. O. Norris of Arthur presented a paper on medical economics. Mrs. J. O. Cletcher entertained the members at the May meeting when Mrs. Philips presented a paper on social insurance and state medicine. The Auxiliary meets whenever Douglas County Medical Society meets. The September meeting was held at the Tuscola Chamber of Commerce. The annual pot-luck luncheon and bridge was held at the home of Dr. and Mrs. Conn in Newman. Mrs. Evans presented a paper at this meeting on "A Doctor's Wife Speaks Up," from the August *Harper's.*

Kane County. The Auxiliary met for dinner with the doctors at the Sherman Hospital, Elgin. Mrs. Solomon Jones was guest of honor and gave a splendid inspirational talk to the ladies after being introduced to

the County Medical Society. She reminded us that human happiness depends upon health, stressed the importance of the work to be done and the responsibility of representing the medical profession. She urged an informed and participating membership. It would be a splendid thing for all Auxiliaries to hear Mrs. Jones and for them to absorb some of the enthusiasm she radiates. The members decided to use a portion of the money on hand to place subscriptions for "Hygeia" in different places in the county. The Auxiliary previously had placed the magazine in the Y. W. C. A., a health center, a library, and a recreation center, where they were very much appreciated. This being the final meeting of the year, Mrs. Imas Rice, the President, gave a resume of the year's work, which included talks by Dr. Eva Wilson, resident physician of Oakdale, the reformatory for women at Dwight; Miss Audrey Campbell, associated with the Illinois Society for the Prevention of Blindness, and Mrs. John T. Mason, member of the Board of the Illinois Society for Prevention of Blindness and largely responsible for the home at Dwight. She talked on "Child Legislation." During the year an advisory committee was appointed by the county medical society; letters and telegrams were sent with their permission on the nitrate bill; a tour of inspection was made through the Copley Hospital at Aurora, and interspersed were two evenings of bridge and a tea. Mrs. Rice then turned the chair over to Mrs. R. C. Hetherington, Geneva, President-Elect, who graciously accepted the office and spoke hopefully of the work to be done. Miss Philis Whitman, clinical psychologist of the Elgin State Hospital, gave an interesting history of the treatment of mental cases from ancient to modern times.

Livingston County. Livingston County Medical Society celebrated its first birthday December 6. Four regular meetings and one special meeting were held during the year, with an average attendance of ten. There were thirty women eligible to membership and fourteen of these belong. The Auxiliary has had round table discussions or speakers at all meetings and has cooperated in sponsoring pre-school examinations in office of physicians. Members have kept posted on medical legislation in Illinois and have written senators or representatives when necessary. The Auxiliary hopes to promote health meetings in women's clubs and to place "Hygeia" in schools as rapidly as funds permit. Books were given to the Livingston County Tuberculosis Sanatorium. At present members are busy sponsoring a Community Food Shower for the benefit of St. James Hospital, which has dispensed an unusual amount of charity during the year. The Auxiliary believes this is an opportunity to interest the public in its work. Non-members are helping and growth in membership is expected, as the drive is county-wide. "We are a small and new organization badly handicapped by lack of funds, but we are striving to keep alive and every member is interested."

McLean County. Mrs. H. W. Grote, President, reports: "Last year we started with only five members. By meeting at the different homes, having supper and a social evening, followed by book reviews and items read from the ILLINOIS MEDICAL JOURNAL, and by each member contributing twenty-five cents, our membership and bank account have been increased. We now have twenty-five members. We have done some philanthropic work and this year see prospects for a much larger membership." Meetings are held the second Tuesday in the month.

Marion County. The Auxiliary to the Marion County Medical Society entertained at a bridge luncheon at Centralia, November 2, in honor of the wives of visiting doctors attending the meeting of the Southern Illinois Medical Association. Forty-three ladies attended the luncheon. In the evening the Auxiliary and visiting ladies were guests at a banquet, following which a special meeting of the Auxiliary was held. The speaker of the evening was Mrs. Philip Kreuscher of Chicago. Her talk was very interesting and pertained to the activities and organization of the Auxiliary. Short talks were also given by Mrs. I. L. Foulon of East St. Louis, Councilor of the 10th District and county president of St. Clair Auxiliary, and Mrs. H. H. Hurd, State Chairman of Hygeia.

St. Clair County. After three months recess the St. Clair County Auxiliary resumed meetings in October. The membership committee is making an effort to add new members and the chairman and president have made eleven calls on prospective members. A successful benefit card party was held on October 28 for the purpose of raising money for Hygeia work in the county. The state chairman of Hygeia is from St. Clair County and the members are all doing their part to aid in her plans. We were very much pleased and honored to have Mrs. Solomon Jones at our November meeting. She gave a very inspiring address to a large gathering of members and wives of doctors of the county. We very much appreciated this visit, for we felt that we were helped by Mrs. Jones' cordial interest and friendly and helpful suggestions. We plan to have a large public relations meeting in February to which we will invite all other clubs of the county.

Rock Island County. An unusually large attendance of members marked the session of the Woman's Auxiliary to the Rock Island County Medical Society in the Rock Island County Tuberculosis Sanitarium, October 9. Mrs. Ruth Mayos, probation officer, spoke most interestingly of her work, and later Mrs. Caroline Juhl, superintendent of the sanitarium, who acted as hostess, served luncheon, assisted by the nurses of the hospital.

Sangamon County. Mrs. E. E. Heglar, President of the Woman's Auxiliary to the Sangamon County Medical Society, gave a tea to the members at the University Club in Springfield on the afternoon of October 6. Dr. John R. Neal gave the address of the afternoon, "The Advantages of the Woman's Auxiliary to the Medical Profession." On November 3, Mrs. Solomon Jones was the honor guest at the regular luncheon and meeting. She gave her intimate experiences in Auxiliary work from the standpoint of the county, state and national.

Vermilion County. Members of the Vermilion County

Medical Society held their monthly meeting at the Danville Woman's Club on November 7 with a dinner followed by an interesting program. Dr. E. G. C. Williams of Danville gave a talk on "Growing Old Gracefully." His discourse was filled with interesting and instructive facts, with a bit of humor about how different people approach the age of maturity. There was lovely music. Some very enlightening facts about the proposed new food and drug act were brought to the members of the Auxiliary by Mrs. O. H. Crist. This bill will probably be brought up before the next congress. Mrs. Jones gave a report of the recent national meeting and outlined the duties and work of the different board members of the state Auxiliary. The annual Christmas party is scheduled for December 5.

Will-Grundy County. The first meeting of the Auxiliary was held November 7 at the home of Mrs. L. J. Frederick, President, at Joliet, with twenty-five present, including three honorary members and three new members. Mrs. Frederick extended greetings and introduced new officers for the year, after which she outlined a program of activities. These include a meeting once a month with the Chairmen of Social, Philanthropy, Legislation, Education and Public Relations presenting programs emphasizing their respective departments. Dr. Bernard Klein, President of the Will-Grundy County Medical Society, addressed the members on the relation of the doctor's wife to her community, ofter which the members retired to the dining room for tea and chatter. The next meeting will be held December 6 at the home of Mrs. John Carey, Chairman of the Philanthropy Committee. This committee will sponsor a "Treasure Chest"—treasures to be toys and books, Christmas gifts for crippled children. The chest will be given to the junior department of the Woman's Club for distribution.

Coles-Cumberland County. The Woman's Auxiliary to the Coles-Cumberland County Medical Society had the State President, Mrs. Solomon Jones, as the speaker at a meeting on September 29. This was held at the home of Mrs. W. R. Rhodes, Toledo. Mrs. Jones gave a most inspiring and helpful talk.

Saline County. Saline County is to have an Auxiliary. All plans are made to have the first meeting on December 19, to be followed by a Christmas party for all doctors and their wives in the county.

NATIONAL BOARD MEETING

A meeting of the Board of the Woman's Auxiliary to the American Medical Association was held in Chicago on October 27. Reports of officers and chairmen were given which indicated progress in organization and plans of work.

EVERY ETHICAL PHYSICIAN IN THE STATE SHOULD BELONG TO THE ILLINOIS STATE MEDICAL SOCIETY

In numbers there is strength. United we stand, divided we fall. A united medical profession can brush away any and all obstacles. It is next to impossible to find a really successful physician who has obtained fame outside the pale of organized medicine.

Are all the eligible physicians in your county members of your local medical society? If nonmembers of local society are discovered get busy and try to induce them to join at the earliest opportunity.

Since the publication of the last edition of the national medical directory, one thousand or more recent graduates and practitioners from other states have located in cities in Illinois. Great numbers have moved within the state from one city to another; an alarming number of physicians have died in the interim; hundreds have moved to other states and a similar number have come into Illinois from other commonwealths.

Below we publish as nearly as possible correct and up-to-date list of physicians residing in towns and cities in Illinois outside of Chicago and Cook County. No attempt had been made to indicate membership in local, state or national medical societies.

The final list of doctors residing outside of Cook County will appear in the forthcoming issue.

ABINGDON (KNOX)
Bradway, Everett Haworth
Meloan, Jos. F.
Rapuzzi, Jos. Edward
Rowe, Jesse
Weir, Clark E.

ADDIEVILLE (WASHINGTON)
Schroeder, Louis P.

ADDISON (DU PAGE)
Brust, Emil Wm.

AKIN (FRANKLIN)
Hamilton, Geo. McClellan

ALBANY (WHITESIDE)
Dimond, Stacy B.

ALBION (EDWARDS)
Apple, Elmer Lawrence
Boston, Andrew Jackson
Brannan, Chas. S.
Moter, Ross Lee

ALEDO (MERCER)
Johnston, Robt. Curry
Mackey, Albert Newton
McClanahan, Victor A.
Moore, Geo. Henry
Rodgers, James Jacob
Sells, Arthur C.
Stites, Hugh Dinsmore
Wallace, James W.

ALEXIS (WARREN)
Crosier, Wm. McKee
Winbigler, Edw. S.

ALGONQUIN (McHENRY)
Beard, Guy Edward
Sculley, Jesse Casstevens

ALLENDALE (WABASH)
Gilliatt, Claude E.
Gray, Frank S.

ALLERTON (VERMILION)
Hardman, Elijah Leonard

ALMA (MARION)
Laswell, Saml. L.

ALPHA (HENRY)
Rothert, Herman

ALSEY (SCOTT)
Bowman, Geo. W.

ALTA (PEORIA)
Allen, Walter H.

ALTAMONT (EFFINGHAM)
Schumacher, Harry Wm.
Stein, Henry
Wright, Chas. M.

ALTON (MADISON)
Baier, Augustus, 1114 Henry St.; office (East Alton)
Bowman, Lincoln M., 1101 E. 5th St.
Brunk, Thos. L., 1633 Washington Ave.
Burnap, Henry Thompson, 2602 College Ave.
Carpenter, Thos. Alan, Alton State Hospital
Carson, Oliver Edward, 2342 State St.; office, 202 E. Broadway
Castle, Stanley, R. D. 2
Cruzat, Roscoe M. (col.), 1916 Gross St.; office, 217A W. 3d St.
Davis, Homer Willard, 312 E. 11th St.; office, 215 E. Broadway
Day, Walter August, 3003 Brown St.; office, 205 W. 3rd St.
Dempsey, Wm. H., 319 Prospect St.; office, 205 W. 3d St.
Eichelberger, Wm. Wirt, Alton State Hospital
Emons, Clifford Wallace, 400 Henry St.; office, 215 E. Broadway
Enos, Edw. Wm., 1917 Beall Ave.; office, Illinois Glass Company
Enos, Wm. H., 325 E. 3d St.
Fiegenbaum, Julius H., 628 Henry St.; office, 308 Belle St.
Fischer, Edward Fredk., 2400 Edwards St.; office, 1524 Washington Ave.
Fisher, Waldo, 503 Summit St.; office, 205 Piasa St.
Giberson, Oria Orton, 538 E. Broadway
Hale, John Peter, 1620 Washington Ave.; office, 200 W. 3d St.
Halliburton, Westley W., 32 W. Broadway
Hastings, James Barnard (5475 Cabanne St., St. Louis); office, 205 W. 3d St.
Hayes, Louis Hodd, 1101 Main St.; office, 620 E. Broadway
Joesting, Fredk. Christian, 1420 Liberty St.; office, 4 W. Broadway
Jones, Fred Wade, 926 Langdon St.; office, 101 E. Broadway
Mayfield, Carey Boyd, Alton State Hospital
McColl, Nettie Iona, Alton State Hospital
McCuistion, Hubert Preston, 2449 Mills Ave.; office, 200 W. 3d St.
McGinnis, Wm. Schlafly, 719 Riverview St.; office, 103 Broadway
Merkle, Clarence Edward, 709 Riverside Dr.; office, 14 E. Broadway
Merkle, Roy Wm., 616 E. 6th St.; office, 14 E. Broadway
Merritt, Nina Polson, 616 3d St.
Middleton, Harry Ernest, 2723 Hillcrest Ave.; office, 103 E. Broadway
Miller, Thos. Wm., 1113 State St.; office, 203 E. Broadway
Morgan, Thos., 2511 State St.
Pfeiffenberger, Mather, 463 Bluff St.; office, 101 E. Broadway
Roberts, Dewey Minnis, 1922 Washington Ave.; office, 614 E. Broadway
Robertson, Alexander Peter, 3029 College Ave.; office, 102 W. 3d St.
Samuels, Geo. Lincoln (col.), 1928 Marilla St.; office, 120 W. 3d St.
Smith, Groves Blake (see Godfrey)
Stewart, James C., Alton State Hospital
Trovillion, Charles E., 2210 Washington Ave.; office, 619 E. Broadway
Vanatta, Clyde Lawrence
Walton, John Edward, 2603 College Ave.
Watson, James Edwin, 2608 State St.
Watson, S. M., 271 Madison Ave.
Wilkinson, Geo. E., 1050 Washington Ave.; office, 209 Alby St.
Williamson, Maurice Rulon, 1411 Main St.; office, 2 E. Broadway
Worden, Geo. Kent, 3107 College Ave.; office, 200 W. 3d St.

ALTONA (KNOX)
Keener, Albert Chester

ALTO PASS (UNION)
Enlow, Chas. Egbert
Stearns, Claude Adams

AMBOY (LEE)
Holladay, Wm. Theodore
Sullivan, Eugene A.
Zeigler, Chas. Arthur

ANNA (UNION)
Benner, Wm. John
Benton, Thos. Jos.
Cohen, Benj.
Eddleman, Wm. Ralph
Goodner, Ralph Allison
Hale, E. Vincent
Hamilton, Angelina G.
Hess, Emory Le Roy
Keith, Leander D.
Keith, Roy
May, Leon Jordan
Nobles, Chas. David
Phillips, Harry
Skorodin, Bernard
Stewart, Don Boyd
Taylor, Harry Otto

ANNAPOLIS (CRAWFORD)
Bartmess, Leonard Ernest

ANNAWAN (HENRY)
Foote, Casper Hartman
Young, James McClain

ANTIOCH (LAKE)
Beebe, Harry Ferris
Deering, David N.
Leslie, Eleanor Isabel
Smith, Halley Ambrose
Warriner, Winsor W.
Williams, Roy D.

APPLE RIVER (JO DAVIESS)
Brink, Chester Abram
Childs, Chas. F.

ARCADIA (R. D. Jacksonville) (MORGAN)
Obermeyer, Albert Everett

ARCOLA (DOUGLAS)
Allen, Elmer Sherman
Barnes, Omer Foutz
Fortney, Millard Hunter
McCain, John H.
Moser, Geo. Henry

ARENZVILLE (CASS)
Crum, Geo. W.
Streuter, Albert Frank

ARGENTA (MACON)
Freeman, Joel C.
Greenfield, Chas. Claud

ARGO (COOK)
Allison, Robt. Howard
Barclay, Robt. Donaldson
Frisbie, Anthony Wayne
Hletko, Paul
Mann, Sidney Louis
Plucinski, Stanislaus Jos.
Rush, Paul White

ARMINGTON (TAZEWELL)
Regnier, Walter Ovid

AROMA PARK (KANKAKEE)
Worstall, Saml. A.

ARROWSMITH (McLEAN)
Johnson, Lyford M.

ARTHUR (DOUGLAS)
McDonald, Chas. Henry
Norris, Clarence Oliver
Phillips, Floyd
Volborn, Albert Lawrence

ASHKUM (IROQUOIS)
Denyes, Orville Leston

ASHLAND (CASS)
Taylor, Walter Sherwood

ASHLEY (WASHINGTON)
Sanders, Claude Jacob

ASHMORE (COLES)
Carrico, Preston O.

ASHTON (LEE)
Calhoun, N. Curtis
Hanawalt, Casper G.

ASSUMPTION (CHRISTIAN)
Coyne, Thos. Arthur
Miller, Wilfred Stare
Rivard, Geo. J.
Zobrist, Benj. Franklin.

ASTORIA (FULTON)
Baxter, Harry Toler
Betts, Wm. Henry
Emerson, Edw. Paul
Fuson, Alonzo Allen
Toler, Thos. W.

ATHENS (MENARD)
Hill, Tolbert F.
Hullerman, Hugo Vincent
Moffett, Wm. Thos.

ATKINSON (HENRY)
Downs, James N.

ATLANTA (LOGAN)
Burke, Chas. Oren
Hutton, Chas. Jesse
Ijams, Russell Lynn
Lee, Maskel

ATWOOD (PIATT)
Veach, Saml. Jos.
Weir, Edgar Wilkin

AUBURN (SANGAMON)
Brittin, Ernest Herbert
Hart, Sterling Perry

AUGUSTA (HANCOCK)
Camp, Julian E.
Cooper, Earl
Wade, Edward H.

AURORA (KANE)
Agnew, Chas. Livington (col.), 653 N. May St.; office, 67 S. Broadway
Allen, Geo. Frank, 149 S. 4th St.
Anderson, James Harlan, 1310 Garfield Ave.; office, 2 N. Broadway
Anderson, Leland Hurd, 749 Oak Ave.; office, 2 S. Broadway
Bailey, Arthur Thos., 50 S. 4th St.; office, 33 Leland Ave.
Balthazar, Eugene Regis, 9 S. Spencer St.; office, 2 S. Broadway
Banker, Edward U., 219 W. Park Ave.; office, 323 Main St.
Bartlett, Fred A., 27 S. Lake St.
Benson, Arnold Sigfrid, 901 S. 4th St.; office, 2 S. Broadway
Boger, Thos. Abram (col.), 1217 Indian Ave.; office, 27 N. Broadway
Bouslough, Elmer E., 411 Spring St.; office, 42 S. Broadway
Brennecke, Herman A., 70 Commonwealth Ave.; office, 323 Main St.
Collins, Richard Kenneth, 253 S. Highland Ave.; office, 323 Main St.
Colwell, Chas. E., 178 S. Lake St.
Coughlin, Frank J., 444 Spring St.; office, 15 S. Broadway
Curtis, Wm. Edward, 111 S. Lancaster Ave.; office, 9 Downer Pl.
Cutter, Cyrus H., R. D. 1
Darmer, Geo. Alex, 922 Downer Pl.; office, 2 N. Broadway
Denney, Elliott Stevenson, 1319 Downer Pl.; office, 33 Island Ave.
Dienst, Richard Carl, 1050 Garfield Ave.; office, 33 Island Ave.
Diller, Adam Elmer, 415 Liberty St.; office, 26 S. River St.
Dreyer, John W., 513 N. Lake St.; office, 33 Island Ave.
Evans, David J., 15 S. View St.; office, 2 N. Broadway
Garrison, Fredk. Franklin, 214 S. Fordham Ave.; office, 2 S. Broadway
Graham, Robt. Hugh, 115 S. Fordham Ave.; office, 323 Main St.
Haan, Geo. W., 425 Marion Ave.; office, 28 Fox St.
Holmblad, Edward Chas.
(See Chicago)
Howard, Irwin Wilson, 61 Le Grand Blvd.; office, 32 S. Broadway
Kaiser, Karl John, 164 S. Gladstone Ave.; office, 30 Island Ave.
Kitenplon, Arthur Nuta, 303 Lawndale Ave.; office, 25 S. Broadway
Klein, Saml., 838 Lebanon St.; office, 4 N. Broadway
Klomhaus, Alfred Paul, Copley Hospital
Lambert, Thos. Jefferson, 423 Blackhawk St.; office, 5 Downer Pl.
Lee, Emmett Lehr, 343 Evans Ave.; office, 2 S. Broadway
Maginnis, Francis Nicholis, 500 N. View St.; office, 10 S. Broadway
McCullough, Jefferson Davis, Jr., 517 S. 4th St.; office, 18 S. Broadway
McLaughlin, Ardsley H., 422 S. 4th St.; office, 30 Island Ave.
Meister, Earl Edward, 630 Talma St.; office, 2 S. Broadway
Milbacher, Walter Herman, 3 S. Lancaster Ave.; office, 33 Island Ave.
Miller, Saml. Douglas III, Copley Hospital
Moore, Albert W., 257 Downer Pl.; office, 33 S. River St.
Murphy, James Orvil, 410 Simms St.; office, 231 N. Broadway
Murphy, John Campbell, 302 Downer Pl.; office, 59 Fox St.
Murphy, Wm. L., 146 Le Grand Blvd.; office, 59 Fox St.
Pritchard, James Eugene, 242 Plum St.; office, 42 S. Broadway
Pulfer, Bernard J., 602 Liberty St.
Putnam, Glenn Worthy, 183 S. 4th St.; office, 33 Island Ave.
Reder, Augustus Ralf, 15 S. May St.; office, 33 Island Ave.
Rice, Imas Pryor, 727 N. Oak Ave.; office, Kane County Spring Brook Sanitarium
Ricker, Sumner J., 149 LaSalle St.; office, 46 S. Broadway
Ritzman, Ralph Roscoe, 338 Lawndale Ave.; office, 67 Fox St.
Robertson, Clarence Wm., 1340 Garfield Ave.; office, 2 S. Broadway
Rogers, Percy Earl, 425 W. Park Ave.; office, 9 Downer Pl.
Rollins, Frank D., 15 S. Lincoln Ave.; office, 115 Main St.
Rudolph, Martin E., Elks Club; office, 33 Island Ave.
Schwingel, Wm. Henry, 231 S. 4th St.; office, 2 S. Broadway
Sherman, Wm. Palmer, 455 N. Lake St.; office, 30 Island Ave.
Stubbs, Edwin Wilfred, R. D. 1
Thomas, Elmer Merill, 736 Grand Ave.; office, 108 Main St.
Turner, Rolland Vincent, 111 Le Grand Blvd.; office, 323 Main St.
Uehren, Wilbur Andrew, 352 N. Lake St.; office, 44 Downer Pl.
Venn, Walter T.
(See Chicago)
Weller, Chas. Grafton, 639 Galena Blvd.; office, 103 Main St.
Westgate, Letitia A., 636 Fox St.
Widmer, Nelson David, 323 Main St.
Wilson, Wm. Wellford, 185 Lincoln Ave.; office, 323 Main St.
Witt, John Desha, 104 Wilder St.
Worthington, David Henry, 569 Fox St.; office, 51 Fox St.
Young, Edmund Valient, 120 Ingleside Ave.; office, 25 S. Broadway

AVA (JACKSON)
Bennett, John
Culp, Theodore W.
Ussery, Raymond E.

AVON (FULTON)
Davis, Ernest E.

BARNHILL (WAYNE)
Simpson, Jos. H.

BARRINGTON (LAKE)
Brooke, Deane Francis
Graber, Benj. Peter
Kleinwachter, Earl Albert
Nelson, Curtis
Weichelt, C. V. Arthur

BARRY (PIKE)
Kaylor, Thos. D.
Kuntz, Wesley W.
Main, Rufus H.

BARTONVILLE (PEORIA)
Borin, Geo. Marion
Borin, Wm. A.
Williams, Herbert Lewis

BASCO (HANCOCK)
Stowe, Israel Fred

BATAVIA (KANE)
Bothwell, Roy Sunderland
Hubbard, Oscar Wilhelm
Mostrom, Henning Theodore
Simon, Oliver Bernard
Spencer, Annie Whitney
West, John Chas.
Zimmerman, Amelia

BATCHTOWN (CALHOUN)
Wood, Harry

BEARDSTOWN (CASS)
Athey, Geo. Lewis
Charles, Thomas Goodell
Garm, Roy Henry
Haworth, Dale Emmett
Holland, Wilbur Wallis
McElroy, Wm. Taylor
Mix, Walter Spaulding
Soule, Chas. Emerson
Vernon, Geo. Heywood

BEASON (LOGAN)
Hollister, Bruce Allan

BEAUCOUP (WASHINGTON)
Neer, David S.

BEAVERVILLE (IROQUOIS)
Anthony, Paul Henry

BEECHER (WILL)
Hiatt, Homer Sam
Kupke, Edward Henry W.
Miley, Michael Ritter
Van Voorhis, Danl. Dewitt

BEECHER CITY (EFFINGHAM)
Parmenter, Geo. Henry

BELLAIR (CRAWFORD)
Ferguson, Robert A.

BELLEVILLE (ST. CLAIR)
Bahrenburg, Wm., 223 E. Main St.
Baldress, Chas. Edward, Jr., 310 S. Charles St.; office, 21 S. High St.
Bauer, Gustav Lewis, Jr., 708 E. Washington St.; office, 7 N. High St.
Baumann, Chas. Hugo, 16 N. Jackson St.
Bechtold, August Fredk., 1305 W. Main St.
Bechtold, Edmond, 3317 W. Main St.; office, N. Illinois St.
Davis, Irvin W., 321 S. Missouri Ave.; office, 116 E. Main St.
Dew, Walter Albert, 513 E. B St.; office, 115a E. Main St.
Duckworth, John Frank, Scott Field
Duey, Delmer R., 25 S. 78th St.; office, 115a E. Main St.
Fisher, Harrison Horton, Scott Field
Frein, Harry Jos., N. Illinois St.; office, 18 W. Main St.
Hansing, Adolph E., 300 N. Illinois St.

Heely, Louis Andrew (St. Libory); office, 9 N. Jackson St.
Heiligenstein, Rudolph C., 11 S. Virginia St.; office, 29 W. Main St.
Hoffmann, John, R. D. 2
Huggins, Chas. R., 517 Park Ave.; office, 10a S. High St.
Huwatcheck, Frank Geo., 100 E. Main St.
Joseph, Raymond John, 6 Country Club Pl.; office, 230 E. Main St.
Kissel, Andrew, 2100 W. Main St.
Lougeay, Saml. McKee, 314 East E St.; office, 230 E. Main St.
Martin, Cecil Lawrence
Massie, John Gore, N. 82nd St.; office, 29 E. Main St.
McKelvey, Saml. Wheeles, 304 Portland Ave.
Meyer, Geo. Edwin, 209 N. 48th St.; office, 20 Public Sq.
Ogle, Oliver Lee, 7301 W. Main St.
Otrich, Grover Cleveland, N. 66th St.; office, 7 N. High St.
Portuondo, Buenaventura H., 203 Abend St.; office, 401 E. Main St.
Raab, Ernst Phillip, 301 S. High St.
Reis, Henry, 110 Wabash Ave.
Renner, Chas. Peter, 120 E. Washington St.; office, 11 S. Church St.
Reuss, Albert Leroy, 315 Wabash Ave.; office, 116a E. Main St.
Roth, Leslie Wm., 3404 W. Main St.; office, 7 N. High St.
Runyon, Wesley Claude, 1819 W. Main St.
Sasville, Ernest Max, R. D. 5
Scruggs, John B., 121 S. Illinois St.
Starkel, Chas. H., 119 S. Charles St.; office, 409 E. Main St.
Stiehl, Elmer Philip, 7 Kircher Pl.; office, 12 West A St.
Twitchell, Benj. E., 107 East D St.; office, 16 S. High St.
Twitchell, James W., 16 S. High St.
Twitchell, Standlee Irving, 505 S. Missouri Ave.; office, 16 S. High St.
Waldman, Jos. Sterling, 100 N. 31st St.; office, 117 E. Main St.
Wangelin, Evans Hugo, 326 S. Pennsylvania Ave.; office, 7 N. High St.
Wheeler, James Earle
Wilson, Clyde Schwerin, 706 Sycamore St.; office, 7 N. High St.
Zepin, David, 1418 W. Main St.; office, 16a N. High St.

BELLMONT (WABASH)

Brian, Chas. Fredk.

BELVIDERE (BOONE)

Alguire, Alden
Alguire, Annie B. McCallum
Dettmann, Everett Fred
Freeman, Wm. M.
Hall, Frank Wilford
Hartman, Minor Leroy
(See Garden Prairie)
Hollnagel, Chas. Wm.
Hood, Kenneth Lindsay
Kaske, Gordon John
Markley, A. J.
McCraken, Clara Bywater
Swift, Arthur W.
West, John Frank
Waitman, Frank S.

BEMENT (PIATT)

Bruner, Allen Maxwell
Burgett, Warren Eugene
McPherson, Warren Gordon

BENLD (MACOUPIN)

Creane, John Chas.
Zerbolio, Dominic Jos.

BENSENVILLE (DU PAGE)

James, Matthew Wm.
Oakes, Fredk.

BENSON (WOODFORD)

Seidl, Ferdinand

BENTON (FRANKLIN)

Alvis, Edw. Carroll
Alvis, Walter Hiram
Buntin, Grover Cleveland
Gore, Geo. Walton
Harris, Monroe
Hart, J. Ottis
Moore, Geo. Gail
Moore, John Beverly
Moore, Jos. E.
Moore, Laurence Moses
Moore, Sydney Clayton
Smith, Wm. H.
Vise, Hosea A.

BERRY (SANGAMON)

Reed, John F.

BETHALTO (MADISON)

Reuter, Walter Jos.
Thrailkill, Jos. S.

BETHANY (MOULTRIE)

Coffey, Roy Calhoun
Donovan, Ophius Poston

BIBLE GROVE (CLAY)

Falley, Richard L.

BIGGSVILLE (HENDERSON)

Babcock, Milton John
Henderson, Wm. D.
Noyes, Frank A.

BIG ROCK (KANE)

Marion, Norman Edw.

BINGHAM (FAYETTE)

Haynes, Moses

BIRDS (LAWRENCE)

Montgomery, John W.

BLANDINSVILLE (McDONOUGH)

MacPherson, Chas. Herbert
Markee, Henry

BLOOMINGTON (McLEAN)

Ball, Orrie Hugh, Jr., 1703 E. Washington St.; office, 219 N. Main St.
Behrendt, Edmund Adelbert, 108 W. Wood St.; office, 211 N. Main St.
Brian, Fredk. Willard, 17 White Pl.; office, 421 N. Main St.
Brown, Edward Lyman, 31 White Pl.; office, 118 N. Center St.
Cantrell, Thos. D., 1201 E. Jefferson St.; office, 310 E. Jefferson St.
Casner, Andrew James, 1501 Clinton Blvd.; office, 120 N. Center St.
Cavins, Lester Blake, 1105 N. Fell Ave.; office, 118 N. Center St.
Cline, Gerald Morris, 1114 Elmwood Rd.; office, 214½ E. Washington St
Condon, John Jos., 1306 N. Main St.; office, 220½ N. Center St.
Deneen, Frank 319½ E. Chestnut St.; office, 219 N. Main St.
Dolley, Homer Odell, 605 Virginia Ave.; office, 421 N. Main St.
Elliott, Jos. Norman, 607 E. Chestnut St.; office, 214½ E. Washington
Fenelon, John H., 311 E. Locust St.; office, 219 N. Main St.
Fielding, Floyd Emerson, 106 Warner Ave.; office, 110 E. Monroe St.
Fisher, Frank C., 608 E. Jefferson St.; office, 219 N. Main St.
Fulwiler, John Walter, 618 E. Walnut St.; office, 211 N. Main St.
Gailey, Watson, Wm. Jr., 5 White Pl.; office, 221 N. Main St.
Grote, Henry Wallace, 505 N. Evans St.; office, 219 N. Main St.
Hart, Edson B., 1314 Fell Ave.; office, 221 N. Main St.
Hartenbower, Geo. Earl (Normal); office, 201 N. Main St.
Hatcher, Wm. Benj. (col.), 1101 W. Washington St.
Hawks, Jos. K. P., 1112 Fell Ave.; office, 219 N. Main St.
Henderson, Frank Harold, 1104 E. Jefferson St.; office, 221 N. Main St.
Howell, Harry Lee, 410 E. Front St.; office, 214 E. Washington St.
Irwin, Lawrence Leslie, 303 E. Jefferson St.; office, 217 N. Main St.
Jones, Clyde Carroll, 1103 E. Jefferson St.; office, 219 N. Main St.
Keller, Alvin, 1223 E. Grove St.; office, Roland Bldg.
Kelso, Geo. Benson, 801 N. Main St.
Leary, Chas. Earl, 105 E. Empire St.
Loar, Ralph Rinehart (Normal); office, 118 N. Center St.
Mammen, Ernest, 303 E. Chestnut St.
Markowitz, Benj., 1108 Elmwood Rd.; office, 219 N. Main St.
Marquis, Vincent Brush, 613 E. Grove St.; office, 219 N. Main St.
McIntosh, James Robt., 214 E. Washington St.
McNutt, James Carson, (Normal); office, 221 N. Main St.
Meyer, Albert W., 606 W. Jackson St.; office, 115½ Front St.
Raber, Daniel Deronda, (Normal); office, 118 N. Center St.
Rhodes, Ora M., 1116 E. Grove St.; office, 101 W. Jefferson St.
Rigg, James R., 1418 N. Clinton Blvd.; office, 118 N. Center St.
Rost, Theodore August, 212 E. Mulberry St.; office, 102 E. Jefferson
Shultz, Chas. Ellis, 509 N. Clinton St.
Sloan, Edwin P., 1417 N. Main St.; office, 219 N. Main St.
Sloan, Guy Arthur, 1703 E. Washington St.; office, 219 N. Main St.
Sloan, Howard Parks, 1501 N. Clinton St.; office, 219 N. Main St.
Small, Geo. H., 601 E. Monroe St.; office, 302 N. Main St.
Smith, Jos. Whitefield, 1122 E. Grove St.; office, 219 N. Main St.
Stevenson, Edgar McLean, 1311 Clinton Blvd.; office, 211 N. Main St.
Watkins, Harold Ryburn, 1319 Clinton Blvd.; office, 221 N. Main St.
Weiland, Edw. E. G., 404 E. Douglas St.

Wellmerling, Hermann Wm., 1411 E. Grove St.; office, 219 N. Main St.
Wikoff, Clarence P., 304 E. Mulberry St.
Winget, Sanford E., 102 W. Front St.
Yolton, Leroy Wm., 208 E. Jefferson St.
Yolton, Rhoda Galloway, 208 E. Jefferson St.
Young, Wm. M., 10 White Pl.; office, 221 N. Main St.

BLUE MOUND (MACON)
Bressmer, Walter Arthur
Matthew, John B.
Montgomery, Clinton Levi

BLUFFS (SCOTT)
Alvarez, Manuel
Evans, Chas. A.

BLUFORD (JEFFERSON)
Ellis, Chas. Lewis
Goodrich, Wm. Ray

BOGOTA (JASPER)
Booker, Carroll

BONE GAP (EDWARDS)
Kitchen, J. Edward
McCormack, James L.

BONFIELD (KANKAKEE)
Schmidli, Charles
Yeates, Wm.

BOWEN (HANCOCK)
Folckemer, Harry Rox
Ketchum, Ellen Pauline
McKim, John B.

BRADFORD (STARK)
Mitchell, Wm. C.
Scholes, James Edward
Terwilliger, Viola E. Shaw

BRADLEY (KANKAKEE)
Goodwin, Andrew J.
(See Kankakee)

BRAIDWOOD (WILL)
Arnold, Romus
(See Joliet)
Frick, Walter Calvin

BREESE (CLINTON)
Morony, James J.
Sauer, Wm. Henry
Warren, Halleck Burkitt

BRIDGEPORT (LAWRENCE)
Fritz, Geo. W.
Lewis, Chas. Marius
Mangum, Wm. Robt.
Schrader, John Frank
Thompson, John Richardson

BRIGHTON (MACOUPIN)
Ash, John Roscoe
Gillham, Anna Merrill
Hall, Thos. Henry
Horine, Thomas A.

BRIMFIELD (PEORIA)
Roberts, Roy B.
Welsh, John Edward

BROADLANDS (CHAMPAIGN)
Dicks, Thos. A.

BROCTON (EDGAR)
Handley, Chas. A.

BROOKPORT (MESSAC)
Gann, Jos. Henry

BROUGHTON (HAMILTON)
Williams, Jos. S.

BROWNS (EDWARDS)
Niemiller, August H.

BRUSSELS (CALHOUN)
Miller, Sam Louis Albert

BRYANT (FULTON)
Axline, Clarence E.

BUCKLEY (IROQUOIS)
Bossart, Harry S.

BUDA (BUREAU)
Taylor, John F.

BUFFALO (SANGAMON)
Black, Orville Evans
Thacker, Edgar Abney

BUFFALO PRAIRIE (ROCK ISLAND)
Marquis, Benj. V.

BUNCOMBE (JOHNSON)
Main, Russell Burton

BUNKER HILL (MACOUPIN)
Bley, Robert E., Jr.
Fuchs, Albert Eugene

BURLINGTON (KANE)
Roach, David Collins

BURNT PRAIRIE (WHITE)
Funkhouser, Robt. M.

BUSHNELL (McDONOUGH)
Griffith, John Curtis
Mahan, Horace Porter, Elmgrove Sanitarium
Rider, Clement J.
Ritchey, George Fenton
Roan, Bert
Roark, John Paul

BYRON (OGLE)
Johnston, James Alba

CABERY (FORD)
Miller, Wm. M.

CAIRO (ALEXANDER)
Barrows, Roy Edgar, Marine Hospital; office, 808 Commercial Ave.
Bondurant, Flint, 2821 Park Pl. W.; office, 800 Commercial Ave.
Chambliss, Homer (col.), 517 24th St.; office, 1312 Poplar St.
Dickerson, Orval Melcher, 701½ Commercial Ave.
Dunn, James Washington, 6th St. and Washington Ave.
Gassaway, James M., 311 5th St.
Hutcheson, Bellenden Seymour, 3003 Park Pl. W.; office, 800 Commercial
Johnson, James Sidney, 2607 Elm St.; office, 800 Commercial Ave.
Lottman, Wm. August (Olive Branch)
McManus, James M., 210 14th St.; office, 808 Commercial Ave.
McNemer, George H., 224 11th St.; office, 608 Commercial Ave.
McNemer, Philip Hamilton, 825 25th St.; office, 608 Commercial Ave.
Miller, Edward Eugene, 2307 Holbrook Ave.; office, 234 8th St.
Rendleman, John J., 2708 Washington Ave.; office, 616 Commercial Ave.
Stuckey, Howard Davis, 1403½ Washington Ave.; office, 708½ Commercial
Weber, Chas. L., 415 Walnut St.; office, 705 Commercial Ave.
Young, Ripley Marion, 3511 Washington Blvd.; office, 1413 Commercial

CALEDONIA (BOONE)
Walgren, Roy Leo

CALHOUN (RICHLAND)
Williamson, Elza L.

CAMBRIDGE (HENRY)
Eaton, Caroline
Eaton, Matilda
Mathre, Albert Ilmer
Westerlund, Jos. E.

CAMDEN (SCHUYLER)
Hayes, Frank Crawford

CAMERON (WARREN)
Zimmerman, Henry Samuel

CAMPBELL HILL (JACKSON)
Busse, Henry Fred

CAMP POINT (ADAMS)
Bates, Amos D.
Martin, Ralph Danl.
Pittman, James Harry

CANTON (FULTON)
Adams, Jasper M., 419 N. 3d Ave.; office, 35 E. Chestnut St.
Betts, Geo. Seymour, R. D. 7
Chapin, Le Roy, 200 Martin Ave.; office, 62 S. Main St.
Coleman, Everett Porter, 224 S. 1st Ave.; office, 24 N. Main St.
Coleman, James Edmund, 656 N. Main St.; office, 24 N. Main St.
Hays, Theodore Calvin, 204 W. Locust St.; office, First National Bank Bldg.
Hays, Verne, 361 W. Pine St.; office, First National Bank Bldg.
Hirschle, Harry Griffith, 245 S. 1st Ave.; office, 26 N. Main St.
Johnston, Cecil James, 150 N. Main St.
Maguire, Roderick Hugh, 121 W. Locust St.; office, 28 W. Elm St.
Nelson, Mark Snowden, 376 W. Chestnut St.; office, 28 W. Elm St.
Putman, Harrison C., 52 N. Avenue D; office, 10 E. Elm St.
Scholes, Paul Sheldon, 372 W. Chestnut St.; office, 74 N. Main St.
Shallenberger, Wm. E., 30 W. Pine St.; office, 80 N. Main St.
Simmons, Jay Claude, 45 S. Avenue B; office, 18 E. Elm St.
Vitt, Louis Leonard, 240 W. Chestnut St.; office, 32 N. Main St.
Vitt, Rose Houda, 240 W. Chestnut St.
Zeigler, Willis Terry, 82 N. Avenue C; office, 26 W. Elm St.

CAPRON (BOONE)
Hutchinson, Geo. Archibald.
Hutchinson, Robt.
Marriett, Woodman R.

CARBONDALE (JACKSON)
Barrow, James Wm.
Bass, Jewell Lee (col.)
Brandon, Wm. Alvin
Brooks, Clyde M.
Caldwell, Delia
Crain, Burt Franklin
Edmondson, Edward Everett
Etherton, Fred Snider
Etherton, Monroe

Hall, Frank Wilburn
Lingle, Fred Lee
Minner, Louis A.
(See Murphysboro)
Moss, Harry Carwin
Rodgers, Emma Washburn

CARLINVILLE (MACOUPIN)
Bell, Robt. H.
Chamness, Earl R.
Denby, John Peter
Liston, Jos. Bostick

CARLOCK (McLEAN)
Hamilton, Chas.

CARLYLE (CLINTON)
DuComb, James Wilson
DuComb, Wilson Lacey
Murphy, Jos. P.
Roane, John Quincy
Wilcox, Saml. E.

CARMI (WHITE)
Brimble-Combe, Wm.
Brown, Raymond Calvin
Crebs, Berry S.
Fahnestock, Edward Ahrens
Legier, John Alfred
Legier, John T.
Sibley, Frank Cassius
Stanley, J. Zeph

CARRIERS MILLS (SALINE)
Davidson, Finis F.
Groce, Robt. E.
Hart, Adiel E.
McSparin, John Monroe.

CARROLLTON (GREENE)
Baldwin, Arthur Kirby
Jouett, Emerit E.
March, Samuel Franklin
Squire, Marguerite G.
Waggoner, Wm. Franklin
Wilson, Arthur Dickinson

CARTERVILLE (WILLIAMSON)
Aird, Andrew John
Boles, Dallas Seth
Coleman, James H.
Fowler, Millard M.
Hiller, Francis M.
Huff, John Preston
Vick, John W.

CARTHAGE (HANCOCK)
Ferris, Chas. Leonard
Frazier, Wilmer Phelps
Runyon, Chas. A.
Scott, Dale Freeland
Sheets, Raymond F.

CARY (McHENRY)
Copeland, Wm. Jackson
Theobald, Frank John

CASEY (CLARK)
Boyd, Rhyne Bashford
Brunker, Herschel Victor
Harris, Cyrus Munson
Johnson, Lester Howard
Lee, I. Wayne

CATLIN (VERMILION)
Dewhirst, Ernest Maxwell
Peel, Chas. Elvie

CAVE IN ROCK (HARDIN)
Gregory, Wm. Grant
Oxford, Seba Ezra

CEDARVILLE (STEPHENSON)
Thompson, Fred Rush
Thompson, Smith C.

CENTRALIA (MARION)
Black, John Earl, 720 E. 3d St.; office, Broadway and Locust St.
Brown, Rudolph H., 814 E. Noleman St.; office, 108½ S. Locust St.
Diehl, Otto Darwin, 400 W. McCord St.; office, 124½ W. Broadway
Edwards, Francis Main, 312½ E. Broadway
Gambill, Wm. Henry, 302 Linden Ave.; office, 138 S. Locust St.
Gillette, Homer Dwight, 401 Leafland Pl.; office, 105 E. Broadway
Hall, John Carroll, 420 Leafland Pl.; office, 102½ E. Broadway
Haney, James M., 104 N. Locust St.
Heller, Augustus Phillip, 128 N. Sycamore St.; office, 102 W. Noleman St.
Holloway, Elza Elvis, 614 W. Broadway; office, 138 S. Locust St.
Kissel, Julius Peter, 315 Linden Ave.; office, Broadway and Locust St.
Marshall, Chas. Pope
Noe, Philip R., 920 E. 3d St.; office, 312½ E. Broadway
Phillips, Francis Ashley, 1403 E. 2d St.; office, 138 S. Locust St.
Plassman, Walter Fredk., 235 S. Hickory St.; office, 105 E. Broadway
Rice, Thos. Warren, 403½ E. Calumet St.
Richardson, Wm. D., 203 N. Locust St.
Stoker, Wm. A., 220 N. Maple St.
Welch, Gilford Nelson, 926 E. 3rd St.; office, 147½ S. Locust St.
Williams, Harry Otto, 423 E. 2nd St.; office, 408 W. 2d St.
Wilson, Harry E., 216 S. Sycamore St.; office, 104 N. Locust St.

CERRO GORDO (PIATT)
Donovan, Herschell V.
Lamb, James Garfield
Trimmer, Albert Ogden

CHADWICK (CARROLL)
Calkins, Arthur Alfred

CHAMPAIGN (CHAMPAIGN)
Allen, Jos. R. (406 W. California Ave., Urbana); office, 1 Main St.
Appelle, Conrad Geo., 1007 W. White St.; office, 206 N. Randolph St.
Armstrong, Ray Conway, 122 W. Hill St.; office, 206 N. Randolph St.
Bach, Irwin Woodward (703 Indiana Ave., Urbana); office, 30 N. Main
Bennett, Cleaves, 712 W. Park St.; office, 113 N. Neil St.
Brewer, Emerson M., 1103 W. Park St.; office, 301 N. Neil St.
Bucher, Clarence Sylvester, 308 W. White St.; office, 209 W. University Ave.
Cavenee, Ebbert LaMonte, 307 Davidson Dr.; office, 113 N. Neil St.
Christie, Chas. Wm., 514 S. Willis St.; office, 44 Main St.
Christie, James Michael, 723 S. Elm St.; office, 44 Main St.
Cleland, James S., 211 E. Healey St.
Colwell, John Bruner, 406 N. State St.; office, 30 Main St.
Craig, Cassius M., 618 W. Hill St.
Dallenbach, John Christian, 1018 W. Church St.; office, 113 N. Neil St.
Dalton, Arthur John, 1019 W. Hill St.; office, 206 N. Randolph St.
Davis, Chas. Summer, 406 W. Springfield Ave.
Dodds, Jos. C., 622 W. Hill St.; office, 76 E. Washington St.
Draper, Edwin Lyon (306 S. Mathews Ave., Urbana); office, 627 S. Wright St.
Eade, Thomas M., 15½ E. University Ave.; office, 1512 W. Springfield Ave.
Ellis, Harry David, 112 N. Walnut St.
Finch, James Hugh, 909 W. University Ave.; office, 113 N. Neil St.
Ford, Hanby Lewis, 812 W. Green St.; office, 206 N. Randolph St.
Garrett, Sherman Scott, 708 W. Church St.; office, 311 W. University
Gernon, Gerald Deland, 406 N. Willis St.; office, 110 N. Neil St.
Gernon, John Henry, 1017 W. Hill St.; office, 113 N. Neil St.
Gray, Wm. L., 211 W. University Ave.; office, 113 N. Neil St.
Greaves, Horatio Norman (202 W. Iowa St., Urbana); office, 28½ Main
Groves, John I., 207 W. Washington St.
Hartford, Wm. Scott, 807 W. Church St.; office, 113 N. Neil St.
Hedgcock, Marcus Whitman, 122 W. Hill St.; office, 44 Main St.
Honn, Wm. M., 311 W. University Ave.
Ingram, Glen Ray, 307 N. Willis Ave.; office, 44 Main St.
Irvin, Cyrus H., 610 W. Church St.; office, 125 W. Church St.
Kariher, Harry Cullen, 712 W. University Ave.; office, 301 N. Neil St.
Kirby, Darwin, 305 W. University Ave.; office, 113 N. Neil St.
Knappenberger, T. Gaillard (801 W. Pennsylvania Ave., Urbana); office, 206 N. Randolph St.
Lamkin, Wm. Franklin, Inman Hotel; office, 209 W. University Ave.
McKinney, Ira, 1002 W. Williams St.; office, 30 Main St.
Miner, Ellen, 209 W. Eureka St.
Moss, Chas. Taylor
(See Urbana)
Newcomb, Cyrus F., 1213 W. Park Ave.; office, 113 N. Neil St.
Osborne, John Wm., 306 W. Clark St.; office, 113 N. Neil St.
Powell, John Rodger, 404 E. White St.; office, 44 Main St.
Rawlings, Harvey Francis (506 S. Matthews Ave., Urbana); office, 113 N. Neil St.
Ross, Vergil Alvin, 1002 W. Springfield Ave.; office, University Health Service
Rowan, Henry Edward (col.), 50 E. Healey St.
Scheib, Geo. F. (1002 S. Lincoln Ave., Urbana); office, 627 S. Wright St.
Scott, Darwin, 804 W. Green St.; office, 113 N. Neil St.
Schowengerdt, Wm. E., 301 E. University Ave.; office, 110 N. Neil St.
Schowengerdt, Wm. Henry, 301 E. University Ave.; office, 110 N. Neil St.

Secker, Wm. V., 806 S. Elm St.; office, 124 N. Neil St.
Shurtz, Richard Chas., 1112 W. Williams St.; office, 44 Main St. and (Thomasboro)
Shurtz, Straut Watson (811 W. Illinois St., Urbana); office, 305 N. Neil St.
Spears, Chas. H., 806 W. Clark St.; office, 113 N. Neil St.
Stilwell, Leland Manford Thos., 206 N. Randolph St.
Sutch, Vincent Jack, 912 W. Williams St.; office, 209 W. University Ave.
West, Jacob Hial, 113 S. Neil St.; office, 116 N. Neil St.
Wilson, Chas. T. C., 509 W. Church St.
Wise, Earl DeWitt, 914 W. Healey St.; office, 113 N. Neil St.
Young, Eugene Yetman, 1016 W. Charles St.; office, 6 Main St.
Zorger, Annie L., 1102 W. Church St.; office, 5 Main St.
Zorger, Wm. H., 1102 W. Church St.; office, 5 Main St.

CHANA (OGLE)
Johnston, Robt. Sherwood

CHANDLERVILLE (CASS)
Boone, Howard B.
Eversole, Garibaldi
Franken, John G.

CHAPIN (MORGAN)
Smith, Lucien

CHARLESTON (COLES)
Alexander, John Richardson
Barnes, Finis E.
Bisson, Martin W.
Bisson, Walter Clinton
Craig, Robt. H.
Dudley, Gerry Brown
Duncan, Chas. Edward
Greer, Chas. Edward
Harwood, Clarence Hugh
Iknayan, Nicholas C.
Lycan, Wm. H.
Oliver, James Alpheus
Shaffer, Henry Albin
Starr, Nathan
Swickard, Clinton Danl.
Swickard, Wm. McHenry

CHATHAM (SANGAMON)
Bradley, Milton Maddox
McLaughlin, Charles
Southwick, Geo. E.

CHATSWORTH (LIVINGSTON)
Palmer, Franklin W.
Seright, Thos. Clinton
Willstead, Otterbein D.

CHAUNCEY (LAWRENCE)
Johnson, Ira Weston

CHEBANSE (IROQUOIS)
Boettcher, Mattie E. Chadwick
Walker, Stanley Ross

CHENOA (McLEAN)
Elfrink, Benj. F.
Kerr, Chas. Roy

CHERRY VALLEY (WINNEBAGO)
Klontz, Chas. Edward

CHESTER (RANDOLPH)
Aszman, Max
Beare, John Wm.
Fritze, Albert E.
Gaines, Quentin McDowell
Hoffman, Geo.
James Wm. A.
Newmark, Israel David
Smith, Louis Jalmer

CHESTERFIELD (MACOUPIN)
Knoop, Wm. A.

CHESTNUT (LOGAN)
Dunn, John Wm.

CHILLICOTHE (PEORIA)
Daugherty, John W.
Smith, Sidney Albert
Thomas, Harry V
Thomas, Ora F.

CHRISMAN (EDGAR)
Conn, Earl Gaines
Huffaker, Columbus
Jennings, James F.
Kerrick, Chas. Lafayette
Scott, Otto Raman
Varner, Robt. Dixon

CHRISTOPHER (FRANKLIN)
Brayfield, Benjamin F.
Dick, Edgar B.
Fieldbrave, Alfred
Odell, Dallas Texas
Rutherford, Orra Leroy
Shafer, Roger Dwight
Sheerer, Walter Winfield

CISCO (PIATT)
Beckstrom-Blanchard, Martha K. J.
Pattengill, Morrell

CISNE (WAYNE)
Bryant, Lawrence K.
Phillips, Wm. Edgar

CISSNA PARK (IROQUOIS)
Roberts, Earl Leslie
Roberts, Wm. Ross

CLAY CITY (CLAY)
Henderson, Curtis

CLAYTON (ADAMS)
Bryant, James Henry
Davis, Floren Fred
Dodd, Frank Clark

CLIFFORD (WILLIAMSON)
Sokoloff, Boris

CLIFTON (IROQUOIS)
Horn, Clinton
Stevens, Nathaniel T.

CLINTON (DEWITT)
Bogardus, Chas. S.
Carter, Chas. W.
Chapin, Clarence W.
Hooker, Henry Kent
Kring, Chas. H.
Marshall, Edward Humphrey
Marshall, Wm. Riddle
Pugh, Bernard McCarthy
Sharkey, Wm. Brady
Shell, Arthur Edwin
Tyler, Aldora J.

COAL CITY (GRUNDY)
Johnson, John Frank
Nicholson, Paul Goodsell

COAL VALLEY (ROCK ISLAND)
Myers, Wm. Fredk.
Myers, Wm. Henry

COATSBURG (ADAMS)
Snider, James Tully

COBDEN (UNION)
Lingle, Willis Edward
Tweedy, James Roy
Wallace, Lesley Edwin

COFFEEN (MONTGOMERY)
Barry, Fredk. W.
McDavid, John L., R. D. 2

COLCHESTER (McDONOUGH)
Dillon, Frank Henry
Harrison, Bruce Ashton

COLFAX (McLEAN)
McIntosh, Wm. B.
Seymour, Guy Emil

COLLINSVILLE (MADISON)
Bellinger, James Edward
Cravens, Harvey G. Mudd
Greaves, Robt. Henry
Harrison, Moses W.
Moore, Eugene Franklin
Oatman, Christopher Lorenzo
Range, Wm. Edgar
Siegel, John H.
Verneuil, Julius Louis

COLUMBIA (MONROE)
Bollinger, Edward
Kohlenbach, Stephen
Lark, Everard T.

COMPTON (LEE)
Owens, Edmund But
Pool, Clarence Gilbert

CONCORD (MORGAN)
Johnson, Augustus M.

COOKSVILLE (McLEAN)
Calhoun, Greer O.

CORDOVA (ROCK ISLAND)
Humphrey, Ira Karr

CORNELL (LIVINGSTON)
Shafer, Harry Lee

COULTERVILLE (RANDOLPH)
Dickinson, Harry Barham
Hendrickson, Geo. Edw.
Kimball, Geo. Wm.
Robertson, John Wylie

COWDEN (SHELBY)
Cherry, Thomas Ewing
Montgomery, Edna Dougherty
Montgomery, Ernest Martin

CREAL SPRINGS (WILLIAMS)
Blanchard, James F.
Glass, Alonzo
Neill, Newman J.

CRESCENT CITY (IROQUOIS)
Kenward, Roy Leslie
Schneider, Ambrose Danl.

CRETE (WILL)
Blim, Chas.
Boyer, John S. (Chicago Heights)
De Alarid, Leon Jess Perez
Kerr, Geo. Austin
Miessler, C. F. Otto

CROPSEY (McLEAN)
Cantle, Herbert Cuthbert

CROSSVILLE (WHITE)
Harper, Augustus Defo

CRYSTAL LAKE (McHENRY)
Alford, Frank Lambert
Lindberg, Alger Victor
Pflueger, Geo. Henry
Sabin, Allen Lucien
Wedge, Athol Horatioa

CUBA (FULTON)
Ewan, Kate E. Armstrong, R. D. 1
Welch, James Wm.

CULLOM (LIVINGSTON)
Dwyer, John Gillman
Robinson, Chas. Hunter

CYPRESS (JOHNSON)
Thomson, Wm.

DAHINDA (KNOX)
Bedford, James Rosser

DAHLGREN (HAMILTON)
Cross, Roland Robert

DAKOTA (STEPHENSON)
Butterfield, Franklin Albert

DALLAS (HANCOCK)
Prescott, Harry V.

DALLAS CITY (HANCOCK)
Cleveland, Austin L.

DALTON CITY (MOULTRIE)
Stevens, Sam. Lorenzo
Wilson, G. Howard
Wilson, Ona Verne

DANA (La SALLE)
Saul, Ora Richard

DANFORTH (IROQUOIS)
Whitsitt, Wilson Henry

DANVERS (McLEAN)
Minnick, Edwin M.
Stickley, Wm. Thos.

DANVILLE (VERMILION)
Allison, Otis Wood, 1017 N. Vermilion St.; office, 9 W. Main St.
Andrews, Perl Keith, 53 S. Virginia Ave.; office, 5 Illinois St.
Arbuckle, Alphonso Taft, 825 E. Fairchild St.
Archibald, James Stewart
Babcock, Henry Stranahan, 110 Robinson St.; office, 106 N. Vermilion St.
Dale, Arthur Ernest, 1645 N. Vermilion St.; office, 106 N. Vermilion St.
Dawson, Dudley Theo., 1121 N. Harmon Ave.; office, 139 N. Vermilion St.
Baldwin, Herschel Edward, 209 Orchard Pl.; office, 106 N. Vermillion St.
Barton, Francis Wm., The Holland; office, 9 W. Main St.
Baumgart, Fredk. August, 924 N. Vermilion St.; office, 9 W. Main St.
Beck, Wm. Rufus King—Veterans Administration Hospital
Becker, Henry F., 6 N. Gilbert St.; office, 106 N. Vermilion St.
Bennett, Corna L., 116 Robinson St.; office, 41 Vermilion St.
Benson, Chas. Prue, National Home for Disabled Volunteer Soldiers
Brandenberger, Arthur Russell, 41 Vermilion St.
Brandt, Ernest H., 24 Pine St.
Caldwell, David Paul, 905 Chandler St.; office, 873 E. Fairchild St.
Carmody, Thos. James, 814 N. Walnut St.; office, 106 N. Vermilion St.
Cass, Geo. Thayer, 1515 E. Main St.; office, 9 W. Main St.
Clements, Robt., 813 N. Gilbert St.; office, 9 W. Main St.
Cloyd, Frazier N., 521 N. Vermilion St.; office, 9 W. Main St.
Cook, Claude Milton, 1216 N. Franklin St.; office, 107½ N. Vermilion St.
Coolley, Elmert Burt, 8 W. Main St.; office, 106 N. Vermilion St.
Cox, Harlan Swarner, 220 E. Woodlawn Ave.
Crispin, Saml. C., 1902 N. Gilbert St.; office, 916 E. Fairchild St.
Crist, Otto H., 1302 E. Main St.; office, 139 N. Vermilion St.
Cunningham, Modie Ezra, 930 E. Main St.
Downs, Henry B., 222 N. Logan Ave.; office, 22 W. North St.
Downs, Maggie Yelton, 222 N. Logan Ave.; office, 22 W. North St.
Dunham, Leslie Herman, 201 E. Conron Ave.; office, 139 N. Vermilion St.
Fairhall, Leo Victor, 1101 Logan Ave.; office, 127½ N. Vermilion St.
Fellows, Wm. Woods, National Home for Disabled Volunteer Soldiers
Fink, Otto Ellsworth, 1122 Sheridan St.; office, 9 W. Madison St.
Fisher, Jacob G., 1014 N. Logan St.; office, 9 W. Main St.
Fithian, Paul H.
Fletcher, Arthur John, 1215 N. Vermilion St.; office, 139 N. Vermilion St.
Funkhouser, Taylor Wilson, 210 W. Roselawn Ave.; office, 106 N. Vermilion St.
Garlitz, Arnold Wm., Veterans Administration Home
Gerety, Wm. Francis, 1712 N. Gilbert St.; office, 126½ E. Main St.
Good, Donald Cameron, 208 W. Dodge Ave.; office, 9 W. Main St.
Good, Richard Warner, Veterans Administration Home
Greenfield, Michael Breinar, National Home for Disabled Volunteer Soldiers
Gregory, Wheeler Russell, National Home for Disabled Volunteer Soldiers
Griswold, Chas. Munson, National Home for Disabler Volunteer Soldiers
Guy, John Milton, 305 W. Seminary St.
Hartsook, Francis Marion, 601 Jewell St.; office, 106 N. Vermilion St.
Hawley, Chas. Lyman K., 101 E. Main St.
Hoffman, Chas. Patrick, 1106 Chandler St.; office, 41 Vermilion St.
Hole, Melvin Leo, 119 Pine St.; office, 106 N. Vermilion St.
Hooker, Henry Frost, Wolford Hotel; office, 9 W. Main St.
Howard, Mordecai Laughlin, 107 Franklin St.
Hundley, James Burnett, 1619 N. Vermilion St.; office, 107½ N. Vermilion St.
Jewell, Benson Mundy, 6 W. Williams St.; office, 106 N. Vermilion St.
Jewell, Earl Bowen, 210 Robinson St.; office, 139 N. Vermilion St.
Johnson, Robt. Elmer, 1424 Oak St.; office, 6 N. Vermilion St.
Jones, Solomon, 604 S. Buchanan St.; office, 106 N. Vermilion St.
King, W. Ivan, National Home for Disabled Volunteer Soldiers
Landauer, Seward L., 1434½ N. Walnut St.; office, 106 N. Vermilion St.
McCaughey, Robt. Stanton, 1112 Sheridan St.; office, 106 N. Vermilion St.
Michael, Oscar James, 507 S. Main St.
Miller, Albert Merrill, 1222 Vermilion St.; office, 106 N. Vermilion St.
Miller, Wm. Rankin, 1414 Oakwood Ave.
Montfort, Roy Melloy, 1004 Lincoln Park Ave.; office, 106 N. Vermilion St.
Moore, Jean Wall, 110 Davidson Dr.; office, 106 N. Vermilion St.
Morgan, Aldine Emmet, National Home for Disabled Volunteer Soldiers
Nigh, John Wesley, National Home for Disabled Volunteer Soldiers
O'Ferrall, Robt. L., 321 Robinson St.
Potter, Geo. Alpha, office, 106 N. Vermilion St.
Rachels, James Harrison, 808 Perrysville Ave.; office, 5½ N. Jackson St.
Ross, Hiram Earl, 1912 Lake Terrace Ave.; office, 9 W. Main St.
Scott, Ira Jos., 614 N. Collett St.; office, 101 N. Vermilion St.
Sherman, Cyril Fantus, Veterans Administration Home
Smith, Warren Braman, 1604 N. Walnut St.; office, 139 N. Vermilion St.
Sorenson, Raymond, National Home for Disabled Volunteer Soldiers
Steiner, Louie Leo, 305 N. Vermilion St.; office, 106 N. Vermilion St.
Van Arsdall, Ernest Payne, 22 N. Pine St.; office, 106 N. Vermilion St.
Walton, Thos. E., 41 N. Vermilion St.
Wellenreiter, Otto Francis, 103 W. Raymond Ave.; office, 106 N. Vermilion St.
Wheatley, Edward James, 1123 N. Logan Ave.; office, 106 N. Vermilion St.
Wilkinson, Charles Edward, 1611 N. Vermilion St.; office, 106 N. Vermilion St.
Williams, Edwin Gordon Culbertson, 308 W. Winter Ave.; office, 106 N. Vermilion St.
Williamson, Carl Syleman, 9 W. Madison Street
Williamson, Holland, 1516 N. Vermilion St.; office, 106 N. Vermilion St.

Williamson, James Holland, 1516 N. Vermilion St.; office, 106 N. Vermilion St.
Wilson, Wm. Hansford, 211½ E. Main St.
Winslow, Edwin Lincoln, 217 W. North St.; office, 41 N. Vermilion St.

DAVIS (STEPHENSON)

Seibert, Henry Hulse
Wilson, Abram Ashley

DECATUR (MACON)

Abrams, Hymen Seelig, Macon County Tuberculosis Sanitarium
Adams, Fred Millard, 1745 N. Church St.
Anderson, Frank M., 600 W. William St.; office, 124 S. Water St.
Arnold, Saml. Edwin, 1203 W. Main St.; office, 124 S. Water St.
Bachrach, Benj., 1555 W. Macon St.; office, 128 E. Main St.
Barnes, Lynn Moore, 124 S. Water St.
Bell, Clarence Elliott, 1336 N. Water St.; office, 250 N. Water St.
Bell, Wm. Henry, 957 N. Water St.
Bohrod, Milton Geo., 1326 W. Decatur St.; office, Decatur and Macon County Hospital
Botts, Alvers T., 1364 N. Church St., office, 301 Water St.
Bourne, Nathan Leander, 904 W. Decatur St.; office, 250 N. Water St.
Burstein, Hymen Jack, 541 N. Front St.; office, 250 N. Water St.
Buxton, Thos. Chas., 903 W. Main St.; office, 239 S. Main St.
Cannon, Vern Edward, 1616 E. William St.
Chandler, Harriet March Day, 1159 W. Decatur St.; office, 132 E. William St.
Chenoweth, Wm. J., 437 W. William St.; office, 129 E. William St.
Collier, Lillie Kiser, 957 W. Wood St.
Curry, Arthur Blythe, 1215 E. William St.; office, 128 N. Main St.
Cussins, James St. Clair, 464 W. Decatur St.; office, 134 E. Prairie St.
Davidson, Wm. Parr, 1147 W. Forrest Ave.; office, 124 S. Water St.
Dudley, Erwin Frank, 1570 W. Forrest Ave.; office, 250 N. Water St.
Eads, Saml. O., 865 W. Main St.; office, 104 N. Water St.
Ellis, John Clinton, 568 S. Haworth St.; office, 339 S. Franklin St.
Faust, John Jabez, 942 N. Main St.; office, 250 N. Water St.
Ferguson, Fred Grant, 340 S. Monroe St.; office, 124 S. Water St.
Fisher, James C., 612 W. Macon St.
FitzPatrick, Martin Wm., 1851 W. Main St.; office, 250 N. Water St.
Flinn, Fauntleroy, 1975 W. Forrest Ave.; office, 220 S. Webster St.
Foster, Alva Curtis, 961 N. Union St.; office, 132 E. William St.
Foster, Thos. Jefferson, 2004 E. Prairie Ave.
Frech, Lee Orville, 1565 W. Macon St.; office, 100 S. Water St.
Garber, Clare Alice, 452 W. Decatur St.; office, 124 S. Water St.
Goodyear, Arthur Fowler, 1398 W. Macon St.; office, 124 S. Water St.
Grissom, Ira Vernon, 421 W. Main St.; office, 124 S. Water St.
Haan, Geo. Wm., Jr., 206 S. Taylor Ave.; office, 226 E. William St.
Harvey, Chas. Francis, 852 W. Wood St.; office, 124 S. Water St.
Hayes, John Maurice, 12 Lincoln Pl.; office, 132 S. Water St.
Hedrick, Wm. Roy, 1715 Main St.; office, 128 E. Main St.
Hopkins, John James, 1183 W. Main St.
Hunt, Augustus Stout, 1343 W. Decatur St.
Jack, Cecil McKee, 451 W. Macon St.; office, 134 W. Prairie Ave.
Johnson, Willard Judd, 906 W. Macon St.; office, 124 S. Water St.
Johnston, Chas. Roy, 1520 Riverview Ave.; office, 250 N. Water St.
Keller, Vincent Fredk., 135 S. Hilton St.; office, 124 S. Water St.
Kennedy, Henry G., 621 S. McClellan St.; office, 124 S. Water St.
Lahners, Thos., Jr., 2215 N. Edward St.; office, 250 N. Water St.
Lindberg, Cosa Dell Haskell, 154 Manor Pl.
Lindberg, David Oscar Nathaniel, 154 W. Court Manor Pl.; office, Macon County Tuberculosis Sanatorium
Long, Vernon M., 2031 N. Edward St.; office, 250 N. Water St.
Lutz, Gustav A., 205 N. Woodlawn Ave.; office, 250 N. Water St.
Magill, Ansel Oswald, 1149 N. College St.; office, 248 N. Water St.
Martin, Forrest Reuben, Macon County Tuberculosis Sanatorium
McClelland, Clarence E., 700 Powers Hill; office, 254 N. Water St.
McClelland, Preston Henkle, 222 Oak Crest
McClelland, Silas Edward, 904 W. William St.; office, 104 N. Water St.
McDavid, Jesse Taylor, 647 S. Crea St.; office, 104 N. Water St.
McGowan, Edwin Chas., 525 N. Franklin Blvd.; office, 146 S. Water St.
McLean, Edwin Philbrook, 224 Park Pl.; office, 124 S. Water St.
Meriweather, Tyler, 646 S. Seigel St.; office, 124 S. Water St.
Mertz, Albert Adolph, 142 S. McClelland Ave.; office, 124 S. Water St.
Miller, John Terrence, 855 W. Wood St.; office, 124 S. Water St.
Murfin, Walter Dean, 312 S. McClelland Ave.; office, 124 S. Water St.
Murphy, Thos. Jos., Wabash Employees' Hospital
Neece, Irving Harrison, 1543 W. Macon St.; office, 250 N. Water St.
Parsons, Herbert E., 2096 W. William St.; office, 1183 W. Main St.
Pence, Dwight A., 250 N. Water St.
Pollock, Milton DeWitt, 6 Montgomery Pl.; office, 250 N. Water St.
Poppele, Oubri A., 904 W. Main St.
Puckett, Francis Leo, 563 S. Broadway; office, Wabash Employees' Hospital
Reeves, Elisha L., 1605 N. Maple St.; office, 140 S. Water St.
Rich, Ciney, 845 W. Forrest St.; office, 250 N. Water St.
Rivard, Geo. Jay, Jr., 637 S. Crea St.; office, 124 S. Water St.
Roos, Edmund Carl, 160 N. Taylor Ave.; office, 134 W. Prairie Ave.
Rose, Milton Edward, 610 S. Monroe St.; office, 134 W. Prairie Ave.
Saling, Wm. J., 224 S. Edward St.; office, 124 S. Water St.
Sanders, Robt. Zink, 626 S. Siegel St.; office, 104 N. Water St.
Simon, Arthur Chas., 853 W. Forest Ave.; office, 250 N. Water St.
Smith, Chas. Raimer, 723 W. Cushing Ave.; office, City Hall.
Smith, Dale DeWitt, 1624 N. Union St.; office, 250 N. Water St.
Smith, Frank Elbert, 504 S. Crea St.; office, 250 N. Water St.
Smith, Stanton Gaston, 1937 E. Williams St.; office, Wabash Employees' Hospital
Spyker, John Huston, 666 S. Siegel St.; office, 104 N. Water St.
Stanley, Dean Field, 224 Home Ave.; office, 250 N. Water St.
Stanley, Otis Orion, 778 W. Decatur St.; office, 250 N. Water St.
Steele, Pierre Abel, 404 W. Macon St.; office, 124 S. Water St.
Strickler, Clarence A., 302 W. Main St.
Strickling, Frank Ellsworth, 1033 W. Wood St.; office, 124 S. Water St.
Talber, Wilmier Melvin, Wabash Employees' Hospital
Tearnan, Clyde Hadrian, 510 Powers Lane; office, 102 N. Water St.
Tearnan, Raymond Arthur, 104 N. Water St.
Thompson, John Clair, 1531 W. Main St.; office, 250 N. Water St.
Trimble, Volantus G., 716 W. Prairie Ave.; office, 124 S. Water St.
Turley, Vigo Thos., 1157 Roger Ave.; office, 916 E. Wood St.
Wente, Constantin Fredk., Decatur and Macon County Hospital
West, Chas. Edward, 145 W. Cerro Gordo St.
White, Justus Vanculen, 1048 W. Macon St.; office, 250 N. Water St.
Wiley, Ralph Emerson, 250 W. Prairie Ave.; office, 124 S. Water St.
Wilhelmy, Orville, 170 S. Dennis Ave.; office, 250 N. Water St.
Wilkinson, Scott Jackson, 1533 W. Main St.; office, 250 N. Water St.
Williamson, Ora Monroe, 228 Woodlawn Ave.; office, 124 S. Water St.
Wilson, Uthie Ray, 354 N. 18th St.; office, 417 N. Water St.
Wood, Chas. Martin, 432 S. Union St.; office, 104 N. Water St.
Wood, Howard M.
(See Mears, Mich.)
Wood, Wilbur C., 605 W. Macon St.; office, 124 S. Water St.
Wood, Wilbur Stuart, 279 Linden Pl.; office, 132 S. Water St.
Woodward, Clayton E., 1070 W. William St.; office, 250 N. Water St.
Yates, Fredk. Hyder, 8 Montgomery Pl.; office, 124 S. Water St.
Yockey, Wm. M., 1006 W. Main St.; office, 540 E. Eldorado St.

DEERFIELD (LAKE)
Davis, Chas. Johnston
Davis, Dorothy Alberta Sugden
Metcalf, Walter Bradford
(See Chicago)
Sugden, Chas. Russell

De KALB (De KALB)
Anderson, Stoddard L.
Badgley, John Anthony
Brown, Mareva Dickerman
Carter, Chas. D.
Cheney, Carl L.
Ellis, James Conrad
Hagey, John B.
Hopkins, Percy Isaiah
Joost, Geo. Herman J.
Rankin, James S.
Rankin, James Tyler
Smith, Clifford Eben
Sundwall, Paul E.
Telford, Elbridge Wright
Wright, Harry Garfield

De LAND (PIATT)
Walker, Geo. Stanberry

DELAVAN (TAZEWELL)
Brink, Harlan W.
Fockler, Geo. W.
Kelchner, Eugene F.
Woltmann, Katharine

De PUE (BUREAU)
Bates, Chas. Richard

De SOTO (JACKSON)
House, Oscar

DETROIT (PIKE)
Goodin, Geo. J.

DEWMAINE (WILLIAMSON)
Springs, Andrew Wilton (col.)

DIETERICH (EFFINGHAM)
Adams, Iduma Ormond
Dimond, Harry Alfred

DIVERNON (SANGAMON)
Johnson, Simeon R.

DIXON (LEE)
Baird, Robt. L.
Brown, Hilles Talley
Chandler, Augustus Wellington
Edwards, Howard Milton
FitzJerrell, Harry Bacon
Garrison, Harriet E.
Glatter, Zoltan M.
Graff, Richard John
Hart, Bruce David
Keator, Louise Helmka (Polo); office, Dixon State Hospital
Lehman, Saml. W.
Lesage, Chas. Herman
Lund, John Mayhue
Marselus, Harry E., Dixon State Hospital
McCoy, Henry James
McNichol, Wm. Arthur
Moore, Amos Foster
Murphy, David Law
Murphy, Edward S.
Murray, Warren Garfield
Parker, Wm. R.
Powell, Curtis F.
Radeff, Ivan Nicholoff
Robbins, Chas. Albert
Segner, Kenyon Bertel
Stackhouse, Stirling Perry
Tarnavsky, Alexander
Thompson, Willard Anthony
Werren, John B.
White, Marion L.

DONGOLA (UNION)
Davis, James Wesley
Parker, Chas. A. C.
Whitaker, Wm. J.

DONNELLSON (MONTGOMERY)
Cary, Alvin Barnard

DONOVAN (IROQUOIS)
Hitchings, Robt. C.

DOWNERS GROVE (DuPAGE)
Bricker, Albert Edwin
Duncombe, Keith Lionel
Ehrler, Glenn Guild
Gourley, Wm. Weir
Gramm, Carl Theodor
Nusbaum, Rayson Lavern
(See Chicago)
Smith, Russell James
Swanson, Gordon Rudolph
Worsley, Edgar Field

DOWNS (McLEAN)
Williams, Edw. C.

DUNDEE (KANE)
Eichler, Theodore Fred
Jones, Archie
Maha, Frank Jos.
Petersen, Heinrich F. W.
Pistor, Ernest M.

DUNLAP (PEORIA)
Wilmot, Clauson Morril

DUPO (ST. CLAIR)
Canaday, Robt. Newton
Cooper, Nevada Costley
Marxer, Barney Jos.

DUQUOIN (PERRY)
Adles, Max
Anderson, Parry Alfred
Brookings, Chas. M.
Bulfer, Andrew Frederic
Byrne, John Dewitt
Carr, Martin C.
Fischer, Geo. Gotthardt
Gutridge, Geo. Harry
Kelly, Thomas Bartlett
Leigh, James T.
Stevens, John Wayne

DURAND (WINNEBAGO)
Lins, Frank Jacob
Roberts, Chas. Alexander

DWIGHT (LIVINGSTON)
Barr, Cyrus H.
Brown, Fredk. Willard
Chadbourne, Arthur Patterson, U. S. Vet. Hospital
Cobb, David Hilliard, Veterans Administration Hospital
George, Raymond Horace, U. S. Vet. Hospital
Joss, Edward Francis
Maupin, Robt. Estill
Neff, Lionel Hubert
Oughton, James Henry
Reynolds, Peter J.
Tripper, Bert
Wilson, Eva McClenahan

EARLVILLE (La SALLE)
Alyea, Thos. E.
Goble, Ezra T.
Lawry, Carl C., R. D. 4
Pierce, Frederick E.
Rayson, Edwin Hope
Wiley, Frank A.

EAST ALTON (MADISON)
Baier, Augustus
(See Alton)
Obert, F. C. Wm.
Quinn, Everette Roy
Toalson, James J.

EAST DUBUQUE (JO DAVIESS)
Lewis, Ulysses Simpson

EAST LYNN (VERMILION)
Vandoren, Raymond Fleming

EAST MOLINE (ROCK ISLAND)
Barding, Lewis Danl., 825 20th Ave.; offices, 1519½ 7th St. and (1630 5th Ave., Moline)
Bollaert, Florens Emma, 1300 18th Ave.
Donahoo, Chas. Edward, 627 16th Ave.; office, 1519½ 7th St.
Fowler, John Henry, 2058 3d Ave.; office, 1509 7th St.
Gamburg, Leo, East Moline State Hospital
Hardinger, Ralph Wilbur, 416 16th Ave.; office, 1509½ 7th St.
Jacobs, Wilhelmina H., East Moline State Hospital
Johnson, Geo. Frederick, 840½ 15th Ave.
Johnston, James Paul, 2344 7th Ave.; office, 912½ 15th Ave.
Koff, Salmon Arthur, East Moline State Hospital
Leff, Abraham, East Moline State Hospital
Lery, Abraham, East Moline State Hospital
Mayos, Chas. Everett, East Moline State Hospital
Solomon, Sam, Jr., East Moline State Hospital
Soule, Earl A., 810 20th Ave.; office, 841½ 15th Ave.
Tackett, Walton, East Moline State Hospital
Timmons, Peter Jos., East Moline State Hospital
Walden Walter
Woodward, Jos. Theodore, East Moline State Hospital

EASTON (MASON)
Russell, Cleveland Clifford

EAST PEORIA (TAZEWELL)
Hopkins, Fred Grant
Stiers, Fredk. Loren
Sullivan, Edward Francis
(See Peoria)

EAST ST. LOUIS (ST. CLAIR)
Alderson, Chas. Franklin, St. Mary's Hospital
Aszmann, Arthur Max, 1926 N. 40th St.; office, 234 Collinsville Ave.
Barker, Roy Frank, 1105 Cleveland Ave.; office, 234 Collinsville Ave.

Barrett, Wm. H. A. (col.); (4313 Enright St., St. Louis); office, 8 S. 4th St.
Beykirch, Jos. Gerhard, Jr., 561 Washington Pl.; office, 234 Collinsville Ave.
Boyd, Olin Blackburn, 1757 Ridge Ave.; office, 403a Collinsville Ave.
Boyd, Tullie Van (Signal Hill); office, 234 Collinsville Ave.
Boyne, Walter Wm., 1663 N. 40th St.; office, 417 Missouri Ave.
Brennan, Michael Earl, 3143 Bond Ave.; office, 513a Missouri Ave.
Bromley, Cerilda Niswonger, 711 N. 11th St.
Brooks, Chas. Newton, 410 Missouri Ave.
Burdick, Geo. Enos, U. S. Vet. Bureau.
Cables, Henry Albert, 529 Washington Pl.; office, 331 Missouri Ave.
Campbell, Richard L., 1100 Summit Ave.; office, 314 Missouri Ave.
Crotty, Wm. John, 641 Vogel Pl.; office, 417 Missouri Ave.
Culbertson, Ora J., 600 Vogel Pl.; office, 234 Collinsville Ave.
De Haan, Adrian John (8300 W. Main St., Belleville); office, 8900 State St.
Donahue, James Jos., 512 Brighton Pl.; office, 327 Missouri Ave.
Doyle, Martin R., 304 N. 10th St.
Ebersole, Sol D., 1402 Summit Ave.; office, 401 Missouri Ave.
Eisele, Chas. Edward, 3806 Caseyville Ave.; office, 300 S. 4th St.
Ellis, Robt. Bertram, 818 N. 24th St.; office, 327 Missouri Ave.
Etherton, John Carroll, 3820 Linden St.; office, 233a Collinsville Ave.
Eubanks, John Edward, Jr., 1433a E. Broadway
Folck, John Kieffer, Christian Welfare Hospital
Foulon, Irenaeus Lister, 608 Washington Pl.; office, 327 Missouri Ave.
Fulgham, John Henry, 515 Washington St.
Green, LaFayette, 3028 Lincoln Ave.; office, 417 Missouri Ave.
Griffith, Wm. Anthony, 1402 Summit Ave.; office, 327 Missouri Ave.
Gunn, Fredk. Henry, 511 Washington Pl.; office, 513a Missouri Ave.
Hanson, Wm. Ludwig, Lindorf Dr.; office, 327 Missouri Ave.
Harney, Louis G., 417 Missouri Ave.
Henry, James Clayton, 3252 Waverly Ave.; office, 234 Collinsville Ave.
Hervey, Wm. Eugene, 1301 St. Clair Ave.
Higgs, John I., 2713 Bond Ave.
Hill, Chas. Edgar, 442 Brighton Pl.; office, 234 Collinsville Ave.
Holten, Edmund Herman, 1621 N. 42d St.; office, 234 Collinsville Ave.
Housh, Ato C., 552 N. 14th St.; office, 417 Missouri Ave.
Hulick, Geo. Oscar, 1533 N. 44th St.; office, 234 Collinsville Ave.
Hunter, Thos. Gerald (col.), 1742 Market Ave.
Hurd, Henry Hill, 1000 Pennsylvania Ave.; office, 431 Missouri Ave.
Isom, Wm. Claiborne, 217 S. Main St.; office, 320a Missouri Ave.
Kane, Clifford Cyrus, 8521 State St.
Killene, Harry Franklin, 1744 N. 23d St.; office, 417 Missouri Ave.
King, Isaiah Horace (col.), 1525 Market Ave.
Kirsch, Francis, 1538 N. 45th St.; office, 303 Collinsville Ave.
Klug, Oscar C., 607 N. 33d St.; office, 417 Missouri Ave.
Knapp, Howard Clay, 705 N. 24th St.; office, 234 Collinsville Ave.
Knewitz, Otto Wm., 1307 N. 13th St.; office, 541 Collinsville Ave.
Le Grand, Danl. W., 463 N. 25th St.
Leon, Fernando, 2757 N. 42d St.
Lippert, John, 1556 St. Clair Ave.
Little, Homer M., 3200 State St.; office, 303 Collinsville Ave.
Little, Finis Coleman, 332 E. Broadway
Little, Rolla E., 1623 State St.; office, 303 Collinsville Ave.
Ludwigs, Gustav Anton John, 921 St. Louis Ave.
Madden, Leo Lawrence, 734 Vogel Pl.; office, 403a Collinsville Ave.
Magarian, Leon, 2525 Caseyville Ave.; office, 1701 Broadway
McCann, Oria Marcellus, 1751 N. 38th St.; office, 234 Collinsville Ave.
McCracken, Robt. X., Granvue Dr.; office, 417 Missouri Ave.
McNary, Wilbert Francis, 424 N. 8th St.; office, 403a Collinsville
McQuillan, Albert Baptiste, Country Club Dr.; office, 327 Missouri
McQuillan, Eugene Albert (Signal Hill); office, 405 Missouri Ave.
Meek, Geo. Chester, St. Mary's Hospital
Menzies, Clifford Gordon (St. Clair Country Club, Belleville); office, 417 Missouri Ave.
O'Brien, Wm. Martin, St. Mary's Hospital
O'Malley, John Jos., St. Mary's Hospital
Rendleman, James W., 553 N. 14th St.; office, 327 Missouri Ave.
Rives, Albert Edgar, 554 N. 18th St.; office, 417 Missouri Ave.
Ryan, Lawrence Augustus, 417 Missouri Ave.
Sharp, Morris Louis, St. Mary's Hospital
Siegel, Vivien Peers, 401a Collinsville Ave.
Skaggs, Chas. Sylvester, 1725 College Ave.; office, 513 Missouri Ave.
Skinner, Geo. Archibald, 801 N. 8th St.
Smith, Harvey S., 2311 Lincoln Ave.; office, 401a Collinsville Ave.
Smith, Henry Devon, 713 St. Louis Ave.
Soucy, John Clovis, 3325 Market St.
Spannagel, Wm. Christian (Signal Hill); office, 137a Collinsville
Spitze, Edw. Christian, 3800 Lincoln Ave.; office, 234 Collinsville
St. John, Dewey, Christian Welfare Hospital
Stanton, Harley Gibson, 755a Collinsville Ave.; office, 1818 N. 40th
Stanton, Roy Francis, 1127 Baugh Ave.
Stines, Thomas Irving, 2310 State St.
Tanzer, Harry Hymen, 614 40th St.; office, 40th St. and Waverly Ave.
Tharp, Royal, 1530 N. 45th St.; office, 327 Missouri Ave.
Thompson, Eugene, 1516 St. Louis Ave.
Triolo, Anthony, St. Mary's Hospital
Twitchell, Robt. A., 611 N. 9th St.; office, 338 Missouri Ave.
Vonnahme, Conrad Benedict, 505 N. 16th St.; office, 228a Collinsville
Voris, Henry McMunn, 1615 N. 45th St.; office, 502a Penn Ave.
Wallace, Thos. Leon, 1436a Piggott Ave.
Waters, Orley Morton, 700 N. 73d St.; office, 225 Collinsville Ave.
Wedel, Louis Emil, 604 Washington Pl.; office, 112a St. Clair Ave.
Wilhelmj, Chas. F. W. (Signal Hill); office, 234 Collinsville Ave.
Wilhelmj, Walter Chas., 518 Missouri Ave.; office, 234 Collinsville
Winning, Chas. Crain, 763 Vogel Pl.; office, 501a Missouri Ave.
Young, John Smith
(See St. Louis)

EDGEWOOD (EFFINGHAM)

Allan, John Gillison

EDGINGTON (R. D. Taylor Ridge) (ROCK ISLAND)

Mosher, Arthur Hall

EDINBURG (CHRISTIAN)

Schott, Wade Hampton
Stokes, Campbell A.

EDWARDSVILLE (MADISON)

Barnsback, Roy Smith
Delicate, Henry Brinkman
Delicate, Wm. Ernest
Ferguson, Edw. C.
Hirsch, Jos. Adams
Monroe, Duncan Danl.
Noggle, Perry L.
(See Granite City)
Oliver Adam Hale
Sutter, John Ritter
Tietze, Herman Christian
Wahl, Eugene, Jr.
Wharff, Howard Eliphalet
Williams, Walter James (Col.)

EFFINGHAM (EFFINGHAM)

Buckmaster, Frank
Burkhardt, Chas. Frederick
Damron, Elbert Leroy
Doty, Chas. M.
Goodell, Frank Wise
Henry, Sylvester Francis
Holman, Clarence C.
Kershner, Jos. Lemuel
Long, Henry August
Reuther, Theodore Ferdinand
Taphorn, D. Henry
Wettstein, John Chas. Robt.

ELBURN (KANE)

Calhoun, Chambers D.
Gates, Wm. S.
Sears, Kenneth Marshall
Taylor, Richard C.

EL DARA (PIKE)

Henry, Gilbert H.

ELDORADO (SALINE)

Beltz, Andrew Hugh
Braun, Wm. E.
Ferrell, Grover C.
Ferrell, John Vernon
Gregory, John H.

Hick, John Carlos
Johnson, Frank
Johnson, Wm. T.
Martin, Cecil Lawrence
Pearce, Franklin Benjamin
Skelton, Neva

ELGIN (KANE)

Abbott, Gordon Wells, 24 S. Liberty St.; office, 165 Milwaukee St.
Allerton, Roy Perry, 5 Douglas Ave.
Azar, Alexander James, 750 S. State St.
Bishop, Wm. H., 18 Hamilton Ave.
Black, Wm. Letcher, Elgin State Hospital
Bridge, Katherine Pleavin, 429 E. Chicago St.
Bridge, Wm. C., 429 E. Chicago St.
Brown, Wm. Simon, 402 N. Spring St.; office, 164 Division St.
Campbell, Peter M., 252 Ann St.; office, 164 DuPage St.
Carpenter, Morgan Gilmore, 402 River Bluff Rd.; office, 165 E. Chicago St.
Carriel, Henry B., 750 S. State St.
Carriel, Joy Ricketts, 750 S. State St.
Chrysanthacopulos, James
(See Chicago)
Collins, Nathan Perry, 337 Jewett St.; office, 161 E. Chicago St.
Cox, Elwood Hunter, Elgin State Hospital
Dewey, Alvin P., 117 N. Liberty St.
Dowell, Raymond Franklin, 864 Highland Ave.; office, 212 Division St.
Dueringer, Henry Wm., 227 Hill St., office, 17 Douglas Ave.
Dunn, Edward Harkless, 557 E. Chicago St.; office, 8 N. Spring St.
Egbert, James K., 398 Chicago St.
Falstein, Eugene Isadore, Elgin State Hospital
Finkelman, Isidore, Elgin State Hospital
Gabby, Saml. Lee, 956 South St.; office, 102 N. Spring St.
Gregg, Wm. Earl, 1039 Logan Ave.; office, 8 N. Spring St.
Griffeth, Frank Wm., 557 E. Chicago St.; office, 8 N. Spring St.
Haffron, Danl. (3911 W. 16th St., Chicago); office, 750 S. State St.
Harcourt, Roy Adolphus, 102 Spring St.
Higgins, Alton J., 514 Spring St.; office, 5 Douglas Ave.
Howell, Sally Yinst, 314 E. Chicago St.; office, 5 Douglas Ave.
Howell, James A. Sullivan, 314 E. Chicago St.; office, 5 Douglas Ave.
Hubrig, Martin Henry, 31 N. Worth Ave.; office, 165 Milwaukee St.
Hughes, Lawrence Jesse, 950 Highland Ave.; office, 163 Milwaukee St.
Hurley, Douglas Chester, 70 S. Chapel St.; office, 171 E. Chicago St.
Jacobs, Milton
(See Chicago)
Jacobson, Jacob Robt., Elgin State Hospital
Kessler, Geo. Brinton, 395 Park St.; office, 8 N. Spring St.
Knight, Howard Talcott, 216 Hamilton Ave.; office, 23 Douglas Ave.
Kocher, Olive Frances Hughes, 559 Parrett St.; office, 16 Spring St.
Krakowski, Julian Peter, 658 Slade
Langhorst, Arthur Louis, 1020 Douglas Ave.; office, 164 Division St.
Langhorst, Fredk. Henry, 600 Chicago St.; office, 164 Division St.
Le Blanc, Florimond Jos., 384 S. Liberty Drive; office, 16 S. Spring
Lescher, Edwin Russell, 1014 N. Spring St.; office, 164 Division St.
Lisor, Graham MacAllister, 364 N. Spring St.; office, 100 E. Chicago St.
Mann, Alban L., 392 E. Chicago St.; office, City Hall
Manougian, Krikore Manoug, 1024 N. Spring St.; office, 164 Division St.
McCornack, Alexander Edwin, 265 Hamilton Ave.; office, 164 Division St.
McIntosh, Jesse Harrison, 750 S. State St.
Milligan, Jay McDonald, 354 Jefferson Ave.; office, 102 N. Spring St.
Mulliken, Oscar Dale, 832 N. Spring St.; office, 5 Douglas Ave.
Nerancy, John Taphy, 750 S. State St.
Pelton, Ora L., 214 S. State St.; office, 102 N. Spring St.
Pillinger, Herbert Henry, 5 Slade Ave.; office, 14 Douglas Ave.
Putnam, Lynn J., 6 S. Gifford St.; office, 102 N. Spring St.
Read, Chas. F., Elgin State Hospital
Reagan, Sheldon Wilson, Elgin State Hospital
Schiller, Maurice Aaron, Elgin State Hospital
Schmidt, Henry Geo. G., 116 S. Chapel St.; office, 8 N. Spring St.
Schneider, Geo. J., 56 N. Channing St.; office, 163 Chicago St.
Schroeder, Mary Gritzner, 750 State St.
Schurmeier, Fredk. Conrad, 849 Douglas Ave.; office, 164 Division St.
Shapiro, Louis Bernard, Elgin State Hospital
Sharp, Chas. E., 685 E. Chicago St.; office, 731 Linden Ave.
Simon, Abraham, Elgin State Hospital
Steinberg, David Louis, 750 S. State St.
Stephens, Wm. Mary, Elgin State Hospital
Tapper, John G., 116 College St.
Tobin, John R., 540 South St.; office, 165 Milwaukee St.
Towner, Francis Robinson, 212 Jefferson St.; office, 102 N. Spring St.
Voight, Benj. John, 716 N. Spring St.; office, 8 Douglas Ave.
Ward, Edna Mae, 750 S. State St.
Weirick, Geo. Albert, 162 S. State St.
Wenner, Henry Lee, Jr., 22 N. Liberty St.; office, 102 N. Spring St.
West, Hugh Hamilton, 56 N. Liberty St.; office, 164 DuPage St.
Wick, Saml., 750 S. State St.
Wilgus, Sidney D.
(See Rockford)
Wiltrakis, Geo. Augustine (4330 Washington Blvd., Chicago); office, Elgin State Hospital
Wittman, Anthony G., Elgin State Hospital
Zapolsky, Irving, Elgin State Hospital

ELIZABETH (JO DAVIESS)

Wiley, Elvin James

ELIZABETHTOWN (HARDIN)

Paris, James Lenard, Sr.; also office (R. D. 1, Karbers Ridge)

ELKVILLE (JACKSON)

Chamness, Clyde James
Harrell, Wm. J.

ELLSWORTH (McLEAN)

Trigger, Harry W.

ELMHURST (Du PAGE)

Abt, A. F. Hugo, 524 S. Hawthorne Ave.
Allen, Robt. A.
Berkey, Harvey Allen
(See Chicago)
Cody, Michael Milton, 204 W. St. Charles Rd.; office, 105 S. York St.
Crane, Milo A.
Elworthy, Robt. Wm., 164 S. York St.
Haney, Irl Stuart
(See Chicago)
Harrison, Wallace Kasson
(See Chicago)
Hills, Lester Harper
Holtz, John Franklin, 135 Prospect Ave.
Horick, Edward Jos., 252 E. 2d St., office, 101 Addison Ave.
Johnson, Richard Geo.
Langhorst, Henry Frederick
Leahy, Frank David, 502 S. Fairview Ave.; office, 532 S. York St.
Marquardt, Edw. Wm.
Mathis, Alvin Leroy, 124 Avon Rd.; office, 107 W. 1st St.
Medley, John W., 177 S. York St.
Scheuerman, Chas. S.
(See Chicago)
Schultz, Harry Louis, 627 Prospect St.; office, 105 S. York St.
Smith, Lorin W., 228 Chandler St.
Tilleman, John Francis, 370 Prospect St.; office, 101 Addison St.
Uthoff, Carl Jos.
(See Oak Park)
Watson, Ernest Starr, 207 W. Fremont St.; office, 105 S. York St.

ELMIRA (R. D. Toulon) (STARK)

Linker, Luther M.

ELMWOOD (PEORIA)

Morton, David Holmes
Varney, Franklin Thos.
Zoll, Edw. C.

EL PASO (WOODFORD)

Gordon, Robt. Earl
McNertney, Frank Daniel
McReynolds, Alva Edgar

ELWOOD (WILL)

Gilbert, Alson R.

EMDEN (LOGAN)

Barringer, Bert Montrose

EMINGTON (LIVINGSTON)

Richardson, Bertram Arthur

ENFIELD (WHITE)

Artin, Arsen Sissak
Staley, Clinton B.

EQUALITY (GALLATIN)

Bourland, Isaac Newton
Womack, James A.

ERIE (WHITESIDE)
Foster, Adin Henry
La Rue, Robt. E.

EUREKA (WOODFORD)
Madison, Wm. Davis
Nickel, Frank Wm.

EVANSVILLE (RANDOLPH)
Beattie, James Glen
Finley, Robt. Linson

EWING (FRANKLIN)
Phillips, John E.

FAIRBURY (LIVINGSTON)
Brewer, Danl.
Langstaff, James Hartzell
Law, Edw. Fredk.
Marshall, Wm. Allen
Thatcher, Allen Horatio

FAIRFIELD (WAYNE)
Blakely, James Thos.
Boggs, John Dalgus
Hancock, Walter Addison
Harlan, James D.
Hilliard, Thos. J.
McDonald, Geo. Albert
Stonemetz, Guy N.
Young, Leslie Winters

FAIRMONT (VERMILION)
Leitzbach, Augustus John

FAIRVIEW (FULTON)
Crouch, Warner Latta
Parks, Doan
Wasson, Redone Edgar

FARINA (FAYETTE)
Holson, James Butler
Phillips, Fredk. Monroe

FARMER CITY (DEWITT)
Hull, Chas. Wilbur
Nowlin, Owen Wendell Emen
Nowlin, Wilfred Jonathan
Ziegler, John H.

FARMERSVILLE (MONTGOMERY)
Hayes, Karl Lowell

FARMINGTON (FULTON)
Dimmitt, Egbert King
Grimm, Ralph Pool
Plumer, Thos. Robt.
(See Trivoli)

FERRIS (HANCOCK)
Kelly, Blair

FIATT (FULTON)
Gray, Wm. Babington

FIELDON (JERSEY)
Brewster, Bert Marion

FILLMORE (MONTGOMERY)
Fish, Wm. Otto

FINDLAY (SHELBY)
Mauzey, Geo. W.

FISHER (CHAMPAIGN)
Donovan, Saml. D.
Sale, Leslie O.

FITHIAN (VERMILION)
Dietrich, Edwin Fredk.

FLANAGAN (LIVINGSTON)
Wilcox, Earl Victor
Zinn, Julian W.

FLAT ROCK (CRAWFORD)
Hardin, Chas. E.
Highsmith, Luther Byrl

FLORA (CLAY)
Bowman, Norton W.
Estes, Clinton Jos.
Fehrenbacher, Harvey Del
Finch, Rollae Danl.
Hutchens, Lucius Lester
Thomas, Wm. Allen

FOREST CITY (MASON)
Blankenship, Clifford R.

FORREST (LIVINGSTON)
Barnhizer, Jay G.
Hamilton, Otis P.

FORRESTON (OGLE)
Akins, Jesse C.
Overfield, Walter W.
Schoon, Theodore Geo.

FORSYTH (MACON)
Lindsey, Lucien Nelson

FORT SHERIDAN (LAKE)
Blair, Lyman Curtis, C. C. Corps
Dibble, John, Station Hospital
Emerson, Gouverneur Vincent, Maj. M.C., U.S.A.
Hayes, Paul, 1st Lieut., M.C., U.S.A.
Hitchens, Arthur Parker, Sixth Corps Area Laboratory
McClintic, Brown Shirk, Maj. M.C., U.S.A.
McDowell, James Rhea, Capt. M.C., U.S.A.
Tarleton, Leeson Oren, Maj. M.C. U.S.A.
Tefft, Wm. H., Col. M.C., U.S.A.

FOUNTAIN GREEN (HANCOCK)
Bouseman, Albert Wm.

FOX LAKE (LAKE)
Rollins, Francis Theodore

FRANKFORT (WILL)
Hedges, Walter Vincent

FRANKLIN (MORGAN)
Henderson, Marion D.
Metcalf, Frederick H.

FRANKLIN GROVE (LEE)
Banker, Frank Marvin

FREEBURG (ST. CLAIR)
Blanchard, Chas. Willard
Dewein, Edward Geo., Jr.
Fox, Fredk. Biggers
Hertel, Henry
Tegtmeier, Chas. Louis

FREEPORT (STEPHENSON)
Ascher, John Alfred, 424 W. Stephenson St.; office, 25½ E. Stephenson St.
Best, Chas. Lorton, 1545 W. Stephenson St.; office, 8½ E. Stephenson St.
Bokhof, David Henry, 518 W. Galena Ave.
Buchanan, James Ned, 123 N. Cherry Ave.; office, 4½ E. Stephenson St.
Burns, Robt. J., 809 S. Liberty St.; office, 4½ E. Stephenson St.
Clark, John Sheldon, 1006 W. Stephenson St.; office, 27 E. Stephenson St.
Diestelmeier, Edward A., 124 N. Harlem Ave.; office, 223 W. Stephenson St.
Grant, John Joseph, 1525 W. Stephenson St.; office, 10 E. Stephenson St.
Harlan, Noah Robt., 1238 W. Stephenson St.; office, 6 W. Main St.
Hartfield, Ernest Monroe, 1060 W. Stephenson St.; office, 12½ E. Stephenson St.
Hewetson, Sara Elizabeth, 868 W. Stephenson St.; office, 4½ E. Stephenson St.
Holke, Theophil J., 1208 W. Stephenson St.; office, 30 W. Stephenson St.
Hutchins, Linda Krape, 638 W. Lincoln Blvd.
Karcher, Wm. L., 1011 W. Stephenson St.; office, 3 W. Stephenson St.
Leavy, Cuthbert Jos., 703 W. Stephenson St.; office, 4½ E St.
Morrison, Hugh Ernest, 115 Foley St.; office, 103 W. Stephenson St.
Motsinger, Ernest Leroy, 1236 S. Maple Ave.; office, 11½ W. Stephenson St.
Peck, Wm. Buckley, 1302 S. Oak Ave.; office, 12½ E. Stephenson St.
Pettepiece, Thomas Arthur, 1039 W. Stephenson St.; office, 222½ W. Stephenson St.
Phillips, Nelson Chancellor, 926 W. Stephenson St.; office, 27 E. Stephenson St.
Poling, James Alonzo, 1439 W. Stephenson St.; office, 202 S. Chicago Ave.
Rideout, Wm. Edward, 1244 W. Stephenson St.; office, 27 E. Stephenson St.
Rieger, Karl Benedict, 424 S. West Ave.; office, 2 E. Main St.
Rosenstiel, Mary L., 6 N. Cherry Ave.; office, 4 N. Cherry Ave.
Shelly, Emanuel Ely, R. D. 5; office, 229 W. Main St.
Sikes, Edward Warren, 1414 W. Stephenson St.; office, 3½ E. Stephenson St.
Snyder, Karl F., La Hacienda; office, 27 E. Stephenson St.
Stickle, Howard John, 1043 W. Lincoln Blvd.; office, 103 W. Stephenson St.
Voigt, Louis George, 415 E. Pleasant St.; office, 27 E. Stephenson St.
Waggoner, Thos. T., 308 W. Main St.
White, Robt. M., 427 S. Carroll Ave.
Zipf, Chas. Bogardus, 1018 W. Douglas St.; office, 27 E. Stephenson St.

FULTON (WHITESIDE)
Durkee, William Henry
Heusinkveld, Henry John
Van de Sand, Gerard F.
Ward, David W.

GALATIA (SALINE)
Empson, M. D.
Garrison, Auda
Grattan, Wm. T. H.

GALENA (JO DAVIESS)
Bench, Edward M.
Dolamore, Jos. F.
Fleege, Francis Henry
Logan, Ray Evan
Schiele, Wm. Christopher

GALESBURG (KNOX)
Augerson, Clara Klopsch, 1638 Bateman St.
Bagley, Henry Patterson, 1549 N. Prairie St.; office, 306 E. Main St.
Baird, Benj. David, 1287 N. Broad St.; office, 64 S. Prairie St.
Birmingham, Thomas F., 591 N. Prairie St.; office, 250 E. Main St.
Bower, Crosiar Geo., 1264 N. Kellogg St.; office, 306 E. Main St.
Bower, Geo. Stuart, 515 N. Chambers St.; office, 306 E. Main St.
Bryant, John Howard, 451 N. Cherry St.; office, 306 E. Main St.
Corbin, John Francis, 135 W. Tompkins St.; office, 152 E. Main St.
Dickinson, Francis Chalffee, 411 N. Prairie St.; office, 64 S. Prairie St.
Eastman, Herbert Clinton, 1198 N. Prairie St.; office, 308 E. Main St.
Fey, Amos, 250 Maple Ave.; office, 64 S. Prairie St.
Finley, Clyde Alexander, 1109 N. Prairie St.; office, 250 E. Main St.
Franing, Edward Chas., 1560 N. Cherry St.; office, 308 E. Main St.
Graham, Howard Edwin, 347 S. West St.; office, 19 E. Main St.
Greenman, John S., 1489 E. Knox St.; office, 64 S. Prairie St.
Griffith, Mose Simpson, 1168 Florence Ave.; office, 250 E. Main St.
Grogan, Edwin Benedict, 247 W. Mary St.; office, 250 E. Main St.
Gunning, Robt. Edward Lee, 219 Walnut Ave.; office, 64 S. Prairie St.
Hall, Fredk. Guy, 624 E. Losey St.; office, 306 E. Main St.
Hill, Columbus C., 429 Johnson St.; office, 152 E. Main St.
Hyslop, Clayton James, 889 N. Broad St.; office, 250 E. Main St.
Johnson, Carl G., 220 N. Chambers St.; office, 306 E. Main St.
Johnson, Nicholas Stanislaus, 820 Florence Ave.; office, 311 E. Main St.
Klein, Grover Cleveland, 1563 N. Cherry St.; office, 311 E. Main St.
Kunz, Frank Oliver, 71 N. Chambers St.; office, Chicago, Burlington and Quincy Railroad Depot
Maley, Geo. Elzear, 390 S. West St.; office, 139 E. Main St.
Maley, Wm. H., 422 N. Cherry St.; office, 44 N. Cherry St.
Matheny, Ralph C., 1030 N. Broad St.; office, 308 E. Main St.
McClanahan, Benj. Vaughn, 969 N. Prairie St.; office, 250 E. Main St.
Murchison, John C., 450 N. Kellogg St.; office, 302 E. Main St.
Nash, Edwin Nelson, 750 N. Academy St.; office, 250 E. Main St.
Pittman, Sanl. M., 86 Phillips St.
Pollock, Arthur David, 971 N. Cherry St.; office, 250 E. Main St.
Quaife, Clarence Edward, 1008 N. Cherry St.; office, 250 E. Main St.
Redington, James Crescent, 1072 Jefferson St.; office, 304 E. Main St.
Rice, Delia M., 1030 N. Broad St.
Ripley, Clarence Bingham, 809 N. Academy St.; office, 250 E. Main St.
Seifert, Edward Herman, 182 N. Prairie St.; office, 249 E. Main St.
Stewart, Alexander Fraser, Galesburg Club
Stotts, Arthur Franklin, 1129 N. Prairie St.; office, 308 E. Main St.
Tate, Louis Neill, 474 N. Academy St.; office, 306 E. Main St.
Toler, John C., 427 N. Prairie St.
Wilbur, Fred M., 1431 N. Prairie St.; office, 250 E. Main St.
Williamson, Wm. S., 450 N. Kellogg St.; office, 18 E. Main St.
Wing, Edgar D., 450 N. Kellogg St.; office, 152 E. Main St.
Winters, Michael Henry, 511 N. Cherry St.; office, 250 E. Main St.
Woods, Silas Samuel (col.), 158 S. Prairie St.
Zessin, Elmer Theo. Paul, 37 E. Grove St.; office, Chicago, Burlington and Quincy Building

GALVA (HENRY)
Alford, John Merlin
Alford, Shirley Foote
Huston, Irvin Everett
Kinsey, Ben W.
Stewart, Robt. Hardy
Waterous, Harry Willard

GARDEN PRAIRIE (BOONE)
Hartman, Minor Leroy

GARDNER (GRUNDY)
Allison, Chas. D.
Fuller, Erlan G.

GARRETT (DOUGLAS)
Colyer, Welby Adams

GAYS (MOULTRIE)
Hardinger, John Daniel

GEFF (WAYNE)
Hilliard, David Alex

GENESEO (HENRY)
Doering, Peter Jos.
Ellingsworth, Jos. Hector
Murphy, John Harvey
Parsons, Arthur
Smith, Robt. Harlie
Spencer, Wilbur F.
Young, Chas. S.
Young, Worling Rivenburg

GENEVA (KANE)
Carpenter, Ralph Waldo
Gray, John Wilson
Harris, Herbert
Hetherington, Robt. Clarke
Knapp, Robt. Dana
Knepper, Paul Albert, Community Hospital
Marstiller, Francis M.
Scott, Raymond G.
Wadsworth, Henry P. (See Chicago)
Weaver, Isabel M. (See Aurora)

GENOA (De KALB)
Baker, Wm. Evan
Burton, Edgar Curtis
Byers, Emery Marcus

GEORGETOWN (VERMILION)
Black, Samuel Milton
Brookshier, Martin L.
Fletcher, Marcus Saml.

GERMANTOWN (CLINTON)
Bauer, John A.
Meirink, Bernard John
Mollet, Emil
Wallace, Robt. Stephen

GIBSON CITY (FORD)
Cunningham, Jos. Swisher
Lane, Robt. Nelson
Potts, Albert LeRoy
Wash, Geo. A.

GIFFORD (CHAMPAIGN)
Axtell, Earl S.

GILLESPIE (MACOUPIN)
Engh, Helmer Arthur
English, John Nelson
Hobson, Edwin Richard Yates
O'Connell, Patrick Benedick
Sullivan, Edward Francis
Thomas, Marvel

GILMAN (IROQUOIS)
Buckner, Roy Alvan
Fordyce, Alexander Wm.
Gainer, John Fisher

GIRARD (MACOUPIN)
Bullard, Ernest Edwin
Finney, Howard Albert
Hill, Green Ewing
Riffey, James Henry

GLADSTONE (HENDERSON)
LoVene, Arthur Wilhelm

GLASFORD (PEORIA)
Yates, Geo. Spurgeon Balshazaar

GLEN CARBON (MADISON)
Schaeffer, Geo. John

GLEN ELLYN (DUPAGE)
Berwanger, Willard Jerome
Camp, Walter John Richard (See Chicago)
Fellows, Fredk. Brown (See Chicago)
Hiatt, Kenneth Nelson
Kay, Abbott Elliott
Kettlestrings, Fred Willis (See Oak Park)
Morrow, John Calvin Weir
Pugh, Cloyd Long
St. Benno, Richard (See Chicago)
St. Benno, Richard W. (See Chicago)
Schiele, Richard Fredk.
Washburne, Geo. F.
Watson, Allen Smith

GODFREY (MADISON)
Smith, Groves Blake

GOLCONDA (POPE)
Barger, Lewis S.
Clark, Thos. H.
Dixon, James W.
Dusch, Lawrence Dale

GOLDEN (ADAMS)
Ross, John Fredk.

GOODFIELD (WOODFORD)
Viers, John Wesley

GOOD HOPE (McDONOUGH)
Bishop, Arthur M.

GOREVILLE (JOHNSON)
Thornton, Alfonzo D.

GORHAM (JACKSON)
Crandle, Eugene

GRAFTON (JERSEY)
Baecht, Frank Chas.
Goitz, Arthur B.

GRAND RIDGE (LASALLE)
Everhart, Arley Glenn
Ives, Augustus

GRAND TOWER (JACKSON)
Baysinger, Millard W.
Gardiner, Chas. D.

GRANITE CITY (MADISON)
Arnovitz, Emanuel Morris, 2501 Delmar Ave.; office, 1916 Delmar Ave.
Binney, Robt. Webster, 19th and D Sts.
Dale, Philip Marshall, 1919 Cleveland Blvd.; office, 1322 Niederinghaus Ave.
Darner, Leslie Dorse, 2561 Cleveland Blvd.; office, 1415 Niederinghaus Ave.
Droege, Edward Henry, 2212 Madison St.; office, 1414, 21st St.
Fitzgerald, James Jos., 2700 Cleveland Blvd.; office, 1908 Edison Ave.
Grayson, Wm. Friar, 1334, 19th St.
Halyama, Gabriel Eugene, 1243a Niederinghaus Ave.
Hamm, Mulfred, 21st and State Sts.
Haven, Wm. Walter, 2040 G. St.
Hoernschemeyer, Jos. Louis, 1332a, 19th St.
Johnson, Frank Oliver, 1938 C St.; office, 1312a Niederinghaus Ave.
Kaylor, Orville Kenneth (See St. Louis)
Krumsiek, Wm. E., 2011 D. St.
Luster, Robt. Dayton, 2337 Cleveland Blvd.; office, 1924 Edison Ave.
Metro, Michael Leo, 2000 E. 19th St.
Noggle, Perry L., (Edwardsville); office, 1635 Niederinghaus Ave.
Phillips, John Henry, 1916 Delmar St.
Purnell, Ernest Alfred, 2509 State St.
Reuss, Harry Pierce, 2257 Cleveland Blvd.; office, 1365a Niederinghaus Ave.
Schermer, Jacob, 2457 State St.; office, 1415 Niederinghaus Ave.
Schreifels, Leonard, 2462 Delmar Ave.; office, 1314a Niederinghaus Ave.
Schroeder, Hugo Christian Herman, 1927 Edison Ave.
Theis, Edward Herman, 2823 Grand Ave.; office, 1365 Niederinghaus Ave.

GRANT PARK (KANKAKEE)
Van Horne, Geo. W.

GRANVILLE (PUTNAM)
Hartman, Perry Vernon
Smart, Clarence Earl

GRAYSLAKE (LAKE)
Anderson, Stanley Danl.
Grim, Ulysses J. (See Chicago)
Pacella, Michelangelo, also office (809 W. Madison St., Chicago)
Struthers, Herbert Rankin

GRAYVILLE (WHITE)
Allison, Hugh Quitman
Bass, Herschel Logan
Green, Ernest A.
Keagy, Cyrus S.

GREAT LAKES (WAUKEGAN P. O.) (LAKE)
Babione, Robt. Wm., Naval Hospital
Belding, Leland James, Naval Hospital
Blew, Clarence Lloyd, Naval Hospital
Boland, Micajah, Naval Training Station
Bradbury, Warren Edwin, Naval Hospital
Dickson, James Gillespie, Naval Hospital
Donelson, Martin, Naval Hospital
Douthat, Robt. Cunningham, Naval Hospital
Edmisten, Loyd Lewis, Naval Hospital
Espach, Walter Clark, Naval Hospital
Harbour, Thos. Quincy, Naval Hospital
Hudson, Millard Fillmore, Naval Hospital
James, Walter Franklyn, Naval Hospital
Johnson, Lewis Wells, Naval Hospital
Judy, Arthur Sinclair, Naval Hospital
Keaney, Felix Patrick, Naval Hospital
Kindleberger, Chas. Poor, Naval Hospital
Leamer, Bruce Vinton, Naval Hospital
Marchand, Decoy, Naval Hospital
Millspaugh, Judson Albert, Naval Hospital
Owen, John Paul, Naval Hospital
Perez, Jose Antonio, Naval Hospital
Raison, Thos. Wrightson, Naval Hospital
Shadday, Alva Adrian, Naval Hospital
Smith, Oliver Augustus, Naval Hospital
Stelter, Emil Jos., Naval Training Station
Storey, Clifford Fulton, Naval Hospital
Vinnedge, Kenneth Houston, Naval Hospital.
Weber, Henry Chas., Naval Hospital
Weinert, Theophilus Franklin, Naval Training Station
Willcutts, Morton Douglas, Naval Hospital
Wood, John Paul, Naval Hospital

GREENFIELD (GREENE)
Bulger, Chas. Oliver
Gause, Orval J.

GREENUP (CUMBERLAND)
Brayshaw, Milton M.
Hancock, Clinton J.
Haughton, Nicholas J.

GREEN VALLEY (TAZEWELL)
No physicians

GREENVIEW (MENARD)
Bowman, Wm. Townzen
Hamil, Chas.
Orr, Loran Ernest
Sprouse, Jennie Glenn

GREENVILLE (BOND)
Cartmell, Harry D.
Cordonnier, Justin Jos.
Cordonnier, Louis Jos.
Dixon, Wm. C.
Easley, Wm. T.
Hall, Wm. Lee
Keith, Archibald M.
Luzader, Katherine Belle
Ravold, Marie L.

GRIDLEY (McLEAN)
Adams, Edwin Melville
Bradford, Mary Elizabeth
Moate, Thos.

GRIGGSVILLE (PIKE)
Chiasson, Placid Nelson
Loveless, Harry Clayton

GURNEE (LAKE COUNTY)
Smith, Winston Wm.
Young, Hiram O. B.

HALLSVILLE (DEWITT)
McLean, Chas. Thos.

HAMILTON (HANCOCK)
Hathaway, Robt. Meredith
Miller, John A.
Runyon, Chas. A. (See Elvaston)

HAMMOND (PIATT)
Lewis, Estelle Vivienne Noe
Lewis, Tullis Blaine
McNutt, John Hestain

HAMPSHIRE (KANE)
Olms, Frank A.

HANNA CITY (PEORIA)
Rohrabaugh, Edward Egleston

HANOVER (JO DAVIESS)
Gollobith, Edward Frank

HARCO (SALINE)
Empson, M. D. (See Galatia)

HARDIN (CALHOUN)
Peisker, Jacob Herman
Skeele, Wm. Amos, Jr.

HARRISBURG (SALINE)
Blackard, Wm. James
Blackard, Wm. James, Jr., 221 W. Poplar St.; office, 2 N. Vine St.
Bond, Robt. Gray, 3 S. Main St.
Burroughs, Edgar Wales, 129 W. College St.; office, 111 E. Poplar St.
Butner, Andrew J., 115 W. Lincoln Ave.; office, Harrisburg Hospital
Butner, Wendell Boise, 2 N. Vine St.
Capel, Jos. V., 121 S. Main St.; office, 2 N. Vine St.
Cummins, Edward W.
Hart, Ezra
Hart, Green B.
Herrmann, Nicholas Anton
Hutton, Byrt Boone
Johnson, Garvies Robinson, 106 E. Lincoln St.; office, Clinic Bldg.
Lehman, Douglas Archer, 105 N. Granger St.; office, 203 N. Vine St.
Lewis, James Henry (col.) 407 E. Walnut St.
Lightner, Jos. Clinton
Lockwood, Kathryn Habermel
Montgomery, Burtis Edgar, 317 W. Poplar St.; office, Lightner Hospital
Nyberg, Robt. Burdett, 203 N. Vine St.
Ozment, Emery Lee
Ozment, Freeman H.
Swan, Walter S., Main and Walnut Sts.
Walden, Chas., 200 W. McHaney St.; office, 111½ N. Main St.

HARTSBURG (LOGAN)
Hutton, Thomas Lee

HARVARD (McHENRY)
Maxon, Jesse Garfield
Royce, Grant Emerson
Schmid, Henry John
Seelye, Norman Lee

HAVANA (MASON)
Auld, Douglas De Vallee
Corey, Fredk. Johnson

Martin, Franklin K.
Steele, Wm. Arthur
Stubenrauch, Chas. Henry

HEBRON (McHENRY)
Bailey, Chas. Whitney
Brown, Edw. V.
Shanahan, Edmond Francis

HECKER (MONROE)
Eckert, Alfred Fred

HEGEWISCH (COOK)
(See Chicago)

HENDERSON (KNOX)
Cooper, Emma Louise

HENNING (VERMILION)
James, Jasper Milton

HENRY (MARSHALL)
Coggeshall, Trovalo Chester
Dysart, Benj. Quincy
Ryder, Bruce Ivan

HERMON (KNOX)
Browning, Arthur L.

HERRICK (SHELBY)
Walker, Glen

HERRIN (WILLIAMSON)
Brown, Columbus, 501 S. 14th St.; office, 201 S. 14th St.
Duncan, Clyde Earl, 817 N. 12th St.; office, 102½ W. Cherry St.
Gardiner, Wm. Robt.
Hennig, Ernst Louis, 102½ W. Cherry St.
Kane, Robt. Leonidas
Lewis, Willis Ivan
Murrah, Frank Clay, 521 S. Park Ave.; office, 201 S. 14th St.
Sanders, Danl. Webster, R. D. 1
Sanders, Francis M., 102½ W. Cherry St.
Tidwell, John Wm., Dell'Era Apts.; office, 216 N. Park Ave.

HERSCHER (KANKAKEE)
Gilborne, Henry Beakley
Wisner, Levi G.

HEYWORTH (McLEAN)
Donovan, Cornelius J.
Hantover, Matthew James
Wakefield, Frank L.

HIGHLAND (MADISON)
Baumann, Nicholas C.
Hermann, Ewald Emil
Kaeser, Albert Frederick
Kempff, Jos. Wm.
Meloy, Earl Stewart
Merwin, Edgar Garfield
Tibbetts, Moses D.
Wirth, Julius

HIGHLAND PARK (LAKE)
Alexander, Harriet C. Beringer, 803 Ravine Dr.
Banfield, Saml. Richard, 2 N. Sheridan Rd.
Bigler, John Adolph, 953 S. Linden Ave.; office, 2 N. Sheridan Rd.
Boyd, Douglas, 2 N. Sheridan Rd.
Byfield, Arthur Fredk. (See Chicago)
Crossman, Roy Arnold, 712 Yale Lane; office, 2 N. Sheridan Rd.
Daggett, Le Roy H. (See Chicago)
D'Wolf, James Francis, 207 Cortland Ave.
Elfrink, Blanche Mayes (See Chicago)
Feingold, Benj. F. (See Glencoe)
Fisher, Edith Tomhagen (See Chicago)
Fitzgerald, James Patrick (See Chicago)
Frank, Ira (See Chicago)
Furno, Peter Hector (See Chicago)
Gatewood (See Chicago)
Gatewood, Lee Connel (See Chicago)
Gradle, Harry Searls (See Chicago)
Grady, Grover Quinton, 529 Forest Ave.; office, 2 N. Sheridan Rd.
Greene, Lois Dixon (See Chicago)
Harkness, Carleton A., 352 Prospect Ave.; office, 2 N. Sheridan Rd.; also office (55 E. Washington St., Chicago)
Herbst, Robt. Harry (See Chicago)
Hinn, Geo. Jacob (See Chicago)
Jacks, Robt. R., 728 Marion Ave.; office, 16 N. Sheridan Rd.
Lackner, Julius Ernest (See Chicago)
Lake, Geo. Burt (See Chicago)
Lundstrom, Jacob Harold (See Highwood)
McNeal, Morley Danl., 839 Lincoln Ave.; office, 2 N. Sheridan Rd.
Meyer, Karl Albert (See Chicago)
Moore, Amory Oliver
Moras, Edmond Raymond, 520 N. Sheridan Rd.
Norcross, Edward Powers (See Chicago)
Norcross, John Ruger, 304 Central Ave.
Roberts, Harry Burton, 330 Elm Pl.; office, 2 N. Sheridan Rd.
Romberger, Arland Seth, Highland Park Hospital
Rossiter, Donald Eugene, 1871 Lyman Ct.; office, 2 N. Sheridan Rd.
Rygh, Edgar Andrew, 275 Cedar Ave.; office, 2 N. Sheridan Rd.
Sheldon, Albert R., Moraine Hotel; office, 2 Sheridan Rd.
Simons, James Danl. (Niles Center); office, 16 N. Sheridan Rd.
Speed, Kellogg (See Chicago)
Teller, Carroll A. (See Chicago)
Tomhagen, Laura Sommer, 914 Ridgewood Dr.
Tremaine, Jay Eugene, 1931 Northmoor Rd.; office, 2 N. Sheridan Rd.
Wagner, Harold Clifford, 318 Oakwood Ave.
Weinfeld, Gustave Frankel (See Ravinia)
Winters, Wm. Lewis, 1409 S. St. Johns Ave.; office, 2 N. Sheridan Rd.

HIGHWOOD (LAKE)
Lundstrom, Jacob Harold (720 W. Park Ave.; Highland Park)
Pearl Allen Sexton, Jr. (626 Skokie Ave., Highland Park)
Risjord, Norman C.
Rossi, Stanislao

HILLSBORO (MONTGOMERY)
Brown, Louis Sylvester
Clotfelter, David Wanless
Douglas, Edmund Turner
Douglas, Wallace Strawn
Hamilton, Robt. Alonzo
Kimball, Zeb Vance
Seymour, Homer A.
Telfer, Geo. Averna

HILLSDALE (ROCK ISLAND)
No physicians

HILLVIEW (GREENE)
Garrison, Harvey W.

HINCKLEY (DE KALB)
Keyes, Arthur Lee
Neubauer, Herman J.

HINDSBORO (DOUGLAS)
Barlow, Roscoe Leland

HINES (COOK)
Allaben, Gerald Randolph, Veterans' Administration Hospital
Anderson, John Birckhead, Edward Hines, Jr., U. S. Vet. Hospital
Barnes, Van Dale, U. S. Vet Bureau
Barrows, Chas. Voyle (2126 S. 5th Ave., Maywood); office, Veterans' Administration Hospital
Biscoe, Frank Lee, Edward Hines, Jr., U. S. Vet. Hospital
Bogen, Eugene Feilen (See Chicago)
Brown, Paul Francis, Edward Hines, Jr., U. S. Vet. Hospital
Cassels, Wm. Geo., Veterans' Administration Hospital
Chapple, James Henry (8 N. 3d Ave., Maywood); office, Edward Hines, Jr., U. S. Vet. Hospital
Christoffersen, Wm. Garrett
Clopton, Abner Theodore, Edward Hines, Jr., U. S. Vet. Hospital
Danziger, Fred (See Chicago)
Davis, Alfred Theophilus (See Chicago)
Day, Wm. Durbin C., Edward Hines, Jr., U. S. Vet. Hospital
Denny, Franklin Thos., Veterans' Administration Hospital
Drew, Henry Cecil (360 Ridge Ave., Evanston); office, Edward Hines, Jr., U. S. Vet. Hospital
Duffy, Frank Thos. (See Chicago)
Ensign, Chas. Francis (1032 S. 12th Ave., Maywood); office, U. S. Vet. Bureau
Etter, Roscoe (2024 S. 4th Ave., Maywood); office, Veterans' Administration Hospital
Feldott, Harry Raymond (See Hinsdale)
Field, Albert L. (See Chicago)
Flynn, John Francis (See Chicago)
Forney, Guy Virl, U. S. Vet. Bureau
Gallagher, Michael Leo (329 Taylor Ave.; Oak Park); office, Edward Hines, Jr., U. S. Vet. Hospital
Gburczyk, Frank Henry (See Chicago)
Gunderson, Elmer Alfred (See Chicago)
Hantsch, Ferdinand Karl (See Chicago)
Hartsaw, John Elmer, Veterans' Administration Hospital
Haslitt, Percy Parker (1414 Maywood Dr., Maywood); office, U. S. Vet. Bureau
Hickman, Harry Edward (See Chicago)
Hollingsworth, Edward West, Edward Hines, Jr., U. S. Vet. Hospital
Hood, John G., Jr. (1018 S. 2d St., Maywood); office, Veterans' Administration Hospital
Howe, John Bell (See Chicago)
Howell, Dent Herman (See Chicago)

Johnstone, Mary M. S. (See Chicago)
Kaplan, David (See Oak Park)
Kesert, Benj. Herman (See Chicago)
Lemning, Howell Elijah (219 N. 2d Ave., Maywood); office, Edward Hines, Jr., U. S. Vet. Hospital
Lewis, James Lang, Edward Hines, Jr., U. S. Vet. Hospital
Little, Edward Orton (See Chicago)
Livingston, Stanton Knowlton, Edward Hines, Jr., U. S. Vet. Hospital
Lyons, Andrew James (Ravinia); office, U. S. Vet. Bureau
Lyons, Clinton Gallagher, Jr., Edward Hines, Jr., U. S. Vet. Hospital
Mallow, Otis Bush, Edward Hines, Jr., U. S. Vet. Hospital
McFadden, Harry Weber (1415 S. 16th Ave., Maywood); office, Edward Hines, Jr., U. S. Vet. Hospital
McNamara, Wm. Lawrence, Veterans' Administration Hospital
Menendez, Anthony Manuel (2040 S. 5th Ave., Maywood)
Miller, D. Herbert (1833 S. 2d Ave., Maywood); office, Veterans' Administration Hospital
Moore, Tarleton Flemming (448 Des Plaines Ave., Forest Park); office, Veterans' Administration Hospital
Mumma, Claude S. (See Chicago)
Murphy, Chas. Percy, Edward Hines, Jr., U. S. Vet. Hospital
Neal, Chas. Allen, Edward Hines, Jr., Veterans' Administration Hospital
Nealon, James Kenneth (1116 Washington Blvd., Oak Park); office, Veterans' Administration Hospital
Palmer, James Price, Veterans' Administration Hospital
Park, Ira Oscar (2004 S. 4th St., Maywood); office, Veterans' Administration Hospital
Perkins, Hanson Travers, Edward Hines, Jr., U. S. Vet. Hospital
Prince, Linnaeus Hodgson, Edward Hines, Jr., U. S. Vet. Hospital
Randolph, Henry Everett (See Chicago)
Reed, Ernest Corydon (205 W. Green St., Maywood)
Scott, Thos. Hugh, Edward Hines, Jr., U. S. Vet. Hospital
Smith, Horace Danl., Veterans' Administration Hospital
Steele, Frank Bell, Edward Hines, Jr., U. S. Vet. Hospital
Sturkey, Edgar Lafayette, Edward Hines, Jr., U. S. Vet. Hosital
Stutz, Wm. Jacob (See Chicago)
Thompson John James, Veterans' Administration Hospital
Torrey, Eugene Weiss, Edward Hines, Jr., U. S. Vet. Hospital
Turner, John Wakeman, Edward Hines, Jr., U. S. Vet. Hospital
Vega, Jaffray Jos., Edward Hines, Jr., U. S. Vet. Hospital
Wallingsford, Wm. Jewell (See Maywood)
Ward, Benj. Franklin (See Cicero)
Weber, Anthony Thos. (See Chicago)
Wegener, Karl Fredk. Ernest, Edward Hines, Jr., U. S. Vet. Hospital

HINSDALE (DUPAGE)

Amacker, Chas. F. (Winfield); office, Natoma Dairy Company
Austin, Margaret Howard (See Chicago)
Bebb, Walter Schuck
Bratrude, Amos Presly
Clowes, Leo Clifford (See Chicago)
Dyas, Fredk. Geo. (See Chicago)
Fiedott, Harry Raymond, also office, (U. S. Vet. Bureau, Hines)
Frank, Wm. Washington
Freeman, Arthur B. (See Chicago)
Lueders, August Henry
Neall, John Howard
Neall, Mary Wild Paulson
Rawlins, Benj. Lewis (See Chicago)
Schultz, Carl Emil
Scott, Ernest Newton
Stuenkel, Arthur John
West, Wm. Kerr
Wohlgemuth, Janet Malcolm
Woodard, Herbert Booth (See Chicago)

HOFFMAN (CLINTON)

Fischer, Andrew Leroy

HOLCOMB (OGLE)

Henderson, Geo. S.

HOMER (CHAMPAIGN)

Brayshaw, Jos.
Johnson, Chas. Edwin
Walton, Jos. Willard

HOOPESTON (VERMILION)

Bell, Jos. Sloan
Brobeck, Alexander Lane
Earel, Albert Marion
Elvidge, Ross Edmund
Hammond, Kenneth Herbert
Johnson, Finley P.
Jones, Leroy
Kline, Ralph Glenn
Moore, Joseph Cleve
Nelms, Chas. Oscar
Russell, Lemuel B.

HOYLETON (WASHINGTON)

Klostermann, Louis J.

HUBBARD WOODS (COOK)

Davis, Nathan Smith III (See Chicago)
Eiseman, Chas., 1278 Scott Ave.; office, 888 Linden Ave.
Fillis, Benjamin Earl
Galloway, Chas. Edwin (See Evanston)
Gay, Robt. James (See Chicago)
Gormley, John Harry,. 1056 Gage St.
Herb, Isabella Coler
Loeb, Clarence
McCarthy, James Edward
Mettler, Lee Harrison
Ruehl, Max C.

HUDSON (McLEAN)

McGee, Geo. R.

HULL (PIKE)

Carder, Lee C.

HUMBOLDT (COLES)

McDougle, James

HUME (EDGAR)

Whalen, Chas. Peter

HUNT (JASPER)

Martin, Lewis

HUNTLEY (McHENRY)

Statler, Oliver Isaiah

HURST (WILLIAMSON)

Burkhart, Victor H.

HUTSONVILLE (CRAWFORD)

Voorheis, Chas. Henry

ILLIOPOLIS (SANGAMON)

Stanford, Vernon Bunn
Willcockson, Homer Bryant

INDIANOLA (VERMILION)

Odbert, Franklin N.
Worthington, Richard R.

INDUSTRY (McDONOUGH)

Botts, Ira Alfred
Stites, Randolph Ora
Stocking, Amer Mills

INGLESIDE (LAKE)

Paden, Chas. M. (See Chicago)

INGRAHAM (CLAY)

Jayne, Wm. J.

IOLA (CLAY)

Cruse, Carl V.

IPAVA (FULTON)

Snively, Chas. Delbert
Stoops, Perry Houston
Wood, Oliver Marshall

IRVINGTON (WASHINGTON)

Klostermann, Geo. Wm.

ITASKA (DU PAGE)

Schroeder, Geo. Fred. C.

IUKA (MARION)

Finn, Walter Louis

IVESDALE (CHAMPAIGN)

Cooper, Wm. Henry

JACKSONVILLE (MORGAN)

Adams, Albyn Lincoln, 871 W. College Ave.; office, 316 W. State St.
Black, Carl Ellsworth, Sr., 1302 W. State St.; office, 2 West Side Sq.
Black, Carl Ellsworth, Jr., 321 Lockwood Pl.; office, 2 West Side Sq.
Bowe, Edward, 134 Park St.; office, 501 W. State St.
Brouse, Ivan Edwin, 316 W. State St.
Buckthorpe, Thelma Ann Cotton, 921 Grove St.
Canatsey, Edward D., 606 N. Church St.; office, 2 West Side Sq.
Clark, Samuel Nye, 1042 Grove St.; office, 1631 W. Mound Ave.
Cole, Chas. E., 1209 W. State St.; office, 234 W. College Ave.
Cravens, James A., 120 S. Main St.
Day, John Ulysses, 918 E. Independence St.; office, 310½ E. State St.
Dewey, Grace, 1123 W. State St.
Dinsmore, Virginia, 303 W. College Ave.
Dollear, Albert H., 1631 Mound Ave.
Doying, Emma Norris, 138 Park St.
Drake, Clarence St. Clair, 1201 S. Main St.
Drennan, Geo. Leland, 1213 W. State St.; office, 2 West Side Sq.
Duncan, Wm. Percy, 356 E. State St.
Engelbach, Hermann Friedrich, 1106 W. State St.
Forkin, Wm. Patrick, 1201 S. Main St.
Frank, Walter Leslie, 244 Park St.; office, 2 W. State St.
Glascoe, Milton Marcellus (col.), 207½ S. Sandy St.

Gregory, Abraham Royden, 551 S. Main St.; office, 2 West Side Sq.
Hairgrove, John Whitlock, 1619 Mound Ave.; office, 2 West Side Sq.
Hardesty, Tully O., 7 Duncan Pl.; office, 336 W. State St.
Hueston, David P., 221 Caldwell St.
Klapman, Jacob W., Jacksonville State Hospital
Lenth, Vincent Thos. James, 2 West Side Sq.
Leschin, Sophie N., 1201 S. Main St.
Marcovitch, Jos., 1201 S. Main St.
McNeely, Celia Alma, 1 Duncan Pl.; office, 2 West Side Sq.
Milligan, Josephine, 1123 W. State St.; office, 2 West Side Sq.
Newcomb, Warner Haines, 1106 W. State St.; office, 316 W. State St.
Norbury, Frank Garm, 1138 W. College Ave.; office, 1631 W. Mound Ave.
Norbury, Frank Parsons (1133 Williams Blvd., Springfield); office, 1631 W. Mound Ave.
Norris, Frank A., 331 W. State St.; office, 2 West Side Sq.
Norris, Reginald Michael, 331 W. State St.; office, 2 West Side Sq.
Patchen, Clayton C., 241 Caldwell St.; office, 237½ W. State St.
Pollock, Fredk. Raymond, Jacksonville State Hospital
Ranes, James L., Jacksonville State Hospital
Reid, David W., 235 W. College Ave.
Ritchie, Chas. Francis P., Jacksonville State Hospital
Roberts, Francis M., 530 W. State St.; office, 317 W. State St.
Schaller, Edward Herbert, 1201 S. Main St.
Weirich, Wm. Harrison, 1609 Mound Ave.; office, 318 W. State St.
Wolever, Aaron P., 1201 S. Main St.
Wolfe, John M., 1309 W. College Ave.; office, 37½ South Side Sq.
Woltman, Henry Clay, 135 Webster Ave.; office, 2 West Side Sq.

JERSEYVILLE (JERSEY)

Bohannan, Hugh Russell
Duguid, Robt. Hurvey
Enos, Laurens
Giers, Louis J.
Gledhill, Henry Robt.
Lewis, Carl Fredk.
Marsden, Bertram A.
Potter, Chas. Chandler

JOHNSTON CITY (WILLIAMSON)

Clayton, John Wesley
Green, Louis H.
Hempler, Herbert Geo.
McKee, John Forsythe
Mozley, John Marshall
Roberts, Ira T.
Tarr, Augustus Willard
Love, Loren Lennoth

Book Reviews

THE SURGICAL CLINICS OF NORTH AMERICA. (Issued serially, one number every other month.) Volume 13, No. 5 (Chicago Number—October, 1933). Octavo of 254 pages with 93 illustrations. Per clinic year, February, 1933, to December, 1933. Paper, $12.00; Cloth, $16.00 net. Philadelphia and London: W. B. Saunders Company, 1933.

The contributors to this number are Doctors Andrews, Bettman, Chandler, Christopher, Compere, Loyal Davis, de Tarnowsky, Herbst, Huggins, Kretschmer, McNealy, Meyer, Miller, Portis, Sarma, Speed and Tivnen.

A MANUAL OF DISEASES OF THE NOSE, THROAT AND EAR. By E. B. Gleason, M. D., LL. D., Professor of Otology, Medico-Chirurgical College Graduate School of Medicine, University of Pennsylvania, Philadelphia. Seventh Edition, Revised and Entirely Reset. 651 pages with 261 illustrations. Philadelphia and London: W. B. Saunders Company, 1933. Cloth, $4.50 net.

In this edition much material has been eliminated, much new material has been added, many paragraphs have been rewritten and there have been many changes throughout the work.

The formulary at the back of the book has been amended to include additional therapeutic details.

A TEXT-BOOK OF MEDICINE (By 141 American Authors). Edited by Russell L. Cecil, A. B., M. D., Sc. D., Professor of Clinical Medicine, Cornell University, Medical College; Associate Attending Physician, New York Hospital, New York City. And Associate Editor for Diseases of the Nervous System, Foster Kennedy, M. D., F. R. S. E., Professor of Neurology, Cornell University, Medical College; Director, Department of Neurology, Bellevue Hospital, New York City. Third Edition, Revised and Entirely Reset. 1664 pages, illustrated. Philadelphia and London: W. B. Saunders Company, 1933. Cloth, $9.00 net.

Since the appearance of the second edition of this work many important advances have been made in internal medicine, especially in the fields of infectious diseases and endocrynology. Several infections have definitely taken their place in the virus-borne group, and important new and fundamental work has broadened our knowledge of the disorders of the pituitary, suprarenal and parathyroid glands. Much new material has been added and the entire book has been reset and the work brought completely up-to-date.

DIET AND DENTAL HEALTH. By Milton T. Hanke. Chicago, Illinois. University of Chicago Press. 1933. Price $1.00.

This work presents detailed information concerning the studies on dental problems that have been carried out in the laboratories of the Otho S. A. Sprague Memorial Institute, University of Chicago, by the Chicago Dental Research club.

NEMBUTAL PREMEDICATION

I use Nembutal, 3 grains (0.194 Gm.) by mouth, in addition to pantopon and scopolamine, for preoperative medication. The pantopon and scopolamine are given one hour before the operation, and Nembutal, 1½ grains, is given a quarter of an hour later and repeated quarter-hourly until the patient is asleep, the maximum dosage being 4½ grains.—Dr. F. P de Caux, Anesthetist Woolwich War Memorial Hospital, London, in *Anesth. & Analg.*, Mar.-Apr., 1932.

ONLY WAITING

Small Boy: "Grandmother, when are you going to start playing football?"

Grandmother: "Why, sonny, I can't play football. Why?"

Small Boy: "Well, papa says he is going to buy a new car as soon as you kick off."—*Carolina Buccaneer.*

Original Articles

THE VETERANS' ORGANIZATIONS AND THE MEDICAL PROFESSION*

F. O. Fredrickson, M. D.
CHICAGO

Mr. Chairman, Colleagues, Members of the Veterans' Service Committee, and Honored Guests: I shall speak to you rather briefly outlining the set-up of the Medical Commission in the Department of Illinois, American Legion, and of the Veterans' Service Committee of the Illinois State Medical Society. Second, I shall attempt to describe to you the veterans' legislation that has been built up in the last ten years and try to lead you up to the passing of the President's Economy Bill. However, I shall not attempt to explain the new veterans' bill which relates to benefits and hospitalization of veterans with service connected disabilities at the present time. The slate has been wiped clean and we are back where we started.

Two years ago the Illinois Department Surgeon of the American Legion introduced a resolution at the Executive Committee of the Department of Illinois recommending that an organization called the Medical Commission be organized. The resolution was adopted. We organized the Medical Commission made up of the Department Surgeon, the Division Surgeons, of which there are five in the state of Illinois, District, County and Post Surgeons. The first year we had about twenty or twenty-five members of this Commission. The following year Dr. Norbury, who was then Department Surgeon, increased the number to about fifty or seventy-five. At the present time we have 100 per cent. Division Surgeons, 5; Districts, 24; Counties, 85; Posts, 189; in other words, we have 303 members of the Medical Commission in the Department of Illinois. Simultaneously with the introduction of this resolution there was a similar resolution introduced into the House of Delegates of the Illinois State Medical Society, recommending the organization of the Veterans' Service Committee. A similar resolution was also introduced in the Council of the Chicago Medical Society recommending a like organization. The object of the Medical Commission is as follows:

First, to effect a close contact between the American Legion and the medical profession through the Veterans' Service Committee of the Illinois State Medical Society. Second, to study carefully all problems of actual interest to the betterment of organized medicine; also to assist service officers of the Legion from a medical standpoint, in proving service connected disabilities in order that cases of deserving veterans may be made compensable. Third, to guide the welfare work of the Legion in such a way as to prevent misunderstandings between the Legion and the County Medical Societies in their respective communities. Fourth, to organize the medical side of the American Legion for effective work in behalf of deserving veterans.

Remember that every case of rehabilitation is a medical problem. I have always advocated that there should be at all times a recognized medical man on the rehabilitation committee of the Legion. So far we have not obtained this. Also, every legionnaire is entitled to the same consideration and same privileges no matter what his profession, be he doctor or lawyer or in other profession or business; in other words, I have in mind that the veteran doctor should receive the same consideration that any other legionnaire or member of any other veteran organization receives.

Fifth, to contact with the Veterans' Service Committee of the County Medical Society in your respective communities.

I should like to present to you an outline of how legislation relative to the veterans' benefits has been brought about. Many things have insidiously crept into these congressional enactments by amendments from time to time. These amendments were often detrimental to the medical profession, not due to the veterans' organizations but due largely to the legislators and congressmen whose only thought has been to be returned to Washington by their constituency.

May I call your attention to the various dates that are important in medical legislation with relation to veterans. Of the three dates, April 6, 1917, is the date war was declared; November 11, 1918, the Armistice was signed and hostilities ceased, and July 2, 1921, is the date when the war closed officially. Around these dates revolved many of the decisions that made for a granting or denial of benefits conferred upon veterans of the World War.

*Read before Veterans' Service Committee Dinner, Peoria, May 16, 1933.

*Statistics Lect. John Bell Howe.

It is impossible to give in detail a comprehensive account of the law upon which the colossal medical function of the Veterans' administration was built up. On October 6, 1917, six months after war was declared, the War Risk Insurance Bureau was organized. This made available to every veteran, life insurance during the war. On August 9, 1921, there was organized the Veterans' Bureau. To this Bureau was transferred all the functions, powers and duties of the War Risk Insurance Bureau, and also there were transferred all the personnel, functions, duties and powers of the Board of Vocational Education, all the property of the U. S. Public Health Service. There was one central office established in Washington, 14 regional offices and 140 sub-offices. All the government facilities were placed at the disposal of the Veterans' Bureau, such as the War and Navy Departments, the Department of the Interior, the Treasury Department and the National Home for Disabled Veteran Soldiers. Also, the Director was instructed to contract state, municipal and private hospitals for the care of veterans. On the above date also the benefits were extended only to veterans of the World War. These disabilities must have occurred in line of duty; in other words, must have been service connected disabilities. Another qualification was that the applicants must have been honorably discharged from the service. Disabilities due to misconduct were entirely uncompensable. Then Section 300 of that law read: Any commissioned officer, enlisted man in the Army or Navy Nursing Corps, injured in line of duty was entitled to benefits or for the aggravation of disabilities that existed at the time of enlistment. The last paragraph in Section 300 relates to a presumptive clause referring to neuro-psychiatric diseases and tuberculosis of the pulmonary type. Under this law those veterans disabled in line of duty were entitled to medical, surgical, convalescent care, dental and prosthetic appliances.

Now then we come to the Congressional Act of April 20, 1922. Bill No. 194 from the 67th Congress which provided for additional hospital construction including all the facilities. There were made available by Congress 17 million dollars. The idea of this Act was to include veterans of other wars in these benefits; such wars as the Spanish-American, the Philippine and Boxer Rebellion and all other expeditions. This, too, related only to neuropsychiatric disease and tuberculosis of the pulmonary type.

On March 4, 1923, Bill No. 542 was passed. This allowed an extension of benefits in line with the presumptive clause that related to tuberculosis irrespective of the organ involved. You can see how gradually the benefits were increasing.

Now then, the Act of 1924, Section 200, extended the benefits to the veterans of all wars, campaigns and expeditions since 1897. On June 7, 1924, there was an extension of the presumptive clause to 1925. Many of these were presumed to have been service connected disabilities. Many of them received the presumption all the way from four to six years. Then there was a further extension in regard to the various diseases, such as tuberculosis, no matter what organ was involved, neuro-osychiatric disease, encephalitis lethargica, and amebic dysentery. You can see that these benefits had been increased from time to time. It was then that the medical profession should have persisted in their protestations against further liberalization of the law. However, legislation went on unrestricted, so that the law relative to those veterans who may receive the benefits was made to include all veterans of all wars and expeditionary forces irrespective of whether they were service connected or not.

Now then, we come to July 3, 1930, the great disability allowance bill. All veterans, irrespective of whether their disability was service connected or not, who had 25 per cent. disability were getting pensions and were permitted to enter veterans' hospitals for treatment. I should like to quote Ray Murphy in the last number of the *Legion Monthly*. He compared this legislation to the Siege of Troy. You all know about the Siege of Troy; when Agamemnon's Greek forces failed to penetrate the impregnable walls of Troy, they built a wooden horse and offered it to the Trojan people. The Trojans accepted it as a gift and dragged it into Troy thinking they were getting something of value. You all know what happened. For the last ten years Congress had been building up a system of veterans' legislation surrounded by an almost impregnable wall of service connection. Finally generous Congress presented the American Legion with a wooden horse in the guise of what we call the disability allowance law. It is this law that caused the destruction of all the veterans' benefits that have

been built up through legislation in the last ten years. So we are back where we started. The slate has been wiped clean. The new Economy Bill, however, created an injustice to certain classes of veterans, particularly with service-connected disabilities. Our President, too, is beginning to realize that such is the case, and no doubt will liberalize the law. As conditions improve there is again the danger of an over-liberalized legislation. Therefore it is for the medical profession to study carefully all future veterans' legislation of mutual interest to the medical profession and the veteran. The medical profession believes that all service connected disabilities should be cared for by the government and that these veterans should be liberally paid for their disabilities and their families who are dependent upon them should be cared for. Another thing, the medical man is always ready to render medical service to deserving veterans unable to pay.

One more point before closing is the question of expenditures. According to General Hines there were $6,318,108,733.00 spent for World War veterans out of a total of $15,370,887,853.00 intended for veterans of all wars during the last 143 years. This is at the rate of $45,774,163.00 per month, of which $26,796,308 went to World War veterans.

In conclusion may I again suggest a continued close cooperation between the Medical Commission of the American Legion, and the Veterans' Service Committee of the Illinois State Medical Society with a view to a definite mutual understanding medically between the medical profession and veterans' organizations.

AMERICAN ACADEMY OF PEDIATRICS IN THE STATE OF ILLINOIS*

GEORGE EDWIN BAXTER, M. D.
CHICAGO

The following quotation describes the organization known as the American Academy of Pediatrics:

The American Academy of Pediatrics is an organization intended to include, as nearly as possible, all of those physicians in the United States and Canada who, by adequate training and proper standards of practice, deserve to be recognized as pediatricians. As Associate Members, it aims to include also those who, by work in allied fields, are able to give it and its members valuable aid in furthering its special objects.

The academy has been formed because of the obvious need of a country-wide organization of pediatricians to perform important services which have not been undertaken by an existing organization.

The objects of the Academy, as stated in its Constitution, are as follows:

Section 1. The object of the Academy shall be to foster and stimulate interest in pediatrics and correlate all aspects of the work for the welfare of children which properly come within the scope of pediatrics. The Academy shall endeavor to accomplish the following purposes:

(a) To establish and maintain the highest standards for pediatric education in medical schools and hospitals, pediatric practice and research;

(b) To perpetuate the history and best traditions of pediatric practice and ethics;

(c) To maintain the dignity and efficiency of pediatric practice in its relationship to public welfare;

(d) To promote publications and encourage contributions to medical and scientific literature pertaining to pediatrics; none of which objects are for pecuniary profit.

It plans actively to further all of these through national, regional and State committees. National and regional scientific meetings will be held. Scientific research and clinical investigation will be encouraged, and publication of worth while studies, monographs, etc., will be facilitated. Standards of education, hospital organization, and practice, will be subjects for constant study.

More than any other branch of medicine, the care of children in its present development is inevitably intimately associated with many social movements which its professors must aid, and often should be prepared to lead and guide, not only as individuals, but through definite organization. The recent reports of the White House Conference have indicated how patent this need is, and how often movements which would better have been under medical guidance have gone on in other hands for lack of available proper medical organization. The Academy is particularly interested in meeting this need satisfactorily. It will make constant study of conditions and progress in all fields of child welfare activity, and endeavor to be prepared through its various committees to furnish competent, authoritative professional aid and guidance wherever these are wanted.

To accomplish these objects, the country has been divided into regions and each region has a certain number of states under its jurisdiction. The Academy operates in each state through the medium of a State Committee. This committee is charged with the task of carrying out in the State the various objects for which the Academy

*Read at Chicago Pediatric Society, October 17, 1933.

is organized. This means that each State Committee shall study the needs of that State and shall formulate a program to meet the conditions present in such State. The committee appointed for the State of Illinois met in the very early spring and formulated a program of activities.

This committee has been energetically at work. The committee brought to the attention of the officers and councilors of the State Medical Society that there was sufficient interest in pediatrics to warrant the establishment of a section on pediatrics.

There was a called meeting in Peoria at the time of the State Medical Society Meeting in May, 1933, inviting Doctors who were interested in the formation of a section on pediatrics. The purpose of that meeting was to determine the sentiment for or against the organization of a section on pediatrics in the Illinois State Medical Society. The subject was presented as outlined in the following statement:

The care of the child is an important part of every physician's practice. The problems in the care of the well child, the diseased conditions in the child, nutrition and growth in childhood, are most important; first, for the sake of the child, and second, for the legitimate betterment of the physician's economic condition. It is necessary for all of us to better inform ourselves in the care of children in order that much of the welfare work now being done by organizations may be done in the Doctor's office.

There has been a rapid and large growth of the specialty of pediatrics. This is evidenced by:

1. A large increase in attendance at the Section on Pediatrics in the American Medical Association, equal to or greater than that in the Section on Medicine.

2. The marked increase in the size of the Chicago Pediatric Society in the past five years. Membership has been more than doubled.

3. The required work in pediatrics in the Class A Medical Schools has about tripled in the past ten years. It is now about equal to the requirements in Gynecology and Obstetrics.

Because of the rapid growth of the specialty of pediatrics, it is necessary to use all available means to bring to the practitioners of medicine the latest advances made in the care of infants and children. The care of infants and children has been popularized with the public to such a degree that numerous organizations have been formed by lay people with popular backing, and in many instances supported professionally by members of our specialty, whose object has been to better child health.

To make the Doctor's office in a community the health center is an ideal if the physician is qualified to meet the demands. There has been created a demand on the part of the public for preventive measures, hygienic teachings, protection against infection and communicable diseases, dietetic management, understanding of behavior problems and disciplinary measures. All of these subjects should be known and taught by the physician as a part of his general medical practice. He should recognize the economic value to the patient as well as to himself. He should know that good reliable medical service is rightfully demanded. He should know how to direct parents in the care of the well child. He has kept himself so much engrossed in the study of disease and pathology that he has neglected the normal physiology and growth factors in infancy and childhood. He has lost or never possessed a child consciousness. Consequently, lay organizations have usurped his rightful place in the health matters of the child. The demand has exceeded the individual Doctor's supply. The physician has failed where lay organizations and trained workers have succeeded. The market has been created for constructive medical service in the growing healthy child and the market has not been supplied with the proper medical supervision. One of the most effective weapons against State Medicine is to be found in the proper care and direction of health problems in the child by the family Doctor.

To accomplish this in the State of Illinois, two things are necessary; first a desire and willingness to teach on the part of those who are qualified; and second, a desire to be taught on the part of those who are not qualified. The post-graduate education of physicians in the subject matter pertaining to the health and the care of children, can be greatly augmented by establishing a section on pediatrics in the State Society, in order to arouse in all physicians a greater child consciousness. We were advised by some who were interested in the formation of a Section that we should put on our own program at the time of a State meeting, and demonstrate to the officers and councilors that there is a real demand for the Section. We found that such a procedure was unnecessary because a sufficient interest was aroused to gain official attention. We were unanimous in the opinion that more could be accomplished by functioning as an integral part of the State Society, than by organizing a State Pediatric Society.

Much to the committee's surprise and gratification, the Council, at the same meeting of the State Society in Peoria, granted the request for a Section on Pediatrics and this Section was to begin to function immediately. The success of this Section on Pediatrics is directly dependent upon the physicians in the State of Illinois who are especially interested in and acquainted with pediatric problems. Our State Society for a number of years has been active in opposing all measures which lead toward State Medicine. Those who have been limiting their work to the

care of children have not sufficiently interested themselves in the work of the State Society. A few men have kept in touch with it but the majority of us have been self sufficient and contented and have utterly failed in our responsibilities to the State Society.

There was the largest gathering of men from our specialty in Peoria that has ever been present at a State Medical Society meeting and I have yet to hear anything but enthusiastic remarks about the good time every one had and a realization that much has been missed in medicine by failure to have become acquainted with the members of the State Society and its activities. It would be well for every member present to read the published report, in the July issue of the ILLINOIS MEDICAL JOURNAL, of the meeting of the House of Delegates. Learn what is actually going on and being done by the members of the State Society. If you know what is being done, you can then give some constructive criticism if you have any.

At the called meeting mentioned above, the following committee was appointed to arrange for the program of the first meeting of the Section on Pediatrics at the State Medical Society Meeting to be held in 1934. Dr. Maurice L. Blatt of Chicago, Chairman; Dr. W. L. Crawford of Rockford, Vice-Chairman and Dr. John R. Vonachen of Peoria, Secretary. This committee was charged with the duty of arranging for the first program to be presented at the opening session of the Section on Pediatrics in the State Society.

Another activity of the State Committee was to appoint a sub-committee whose duty it is to contact with all governmental agencies throughout the State for the purpose of aiding or advising with governmental agencies in all matters pertaining to the welfare of children. This contact has been made and two members, Clifford Grulee and Sam Hoffman, of the Academy, have been appointed on the advisory committee. This committee is headed by an appointee from the State Department of Health.

A third activity was the appointment of a committee to contact with various lay organizations throughout the City and State for the purpose of advising with the members of these organizations and attempt to direct their activities from a medical standpoint. These contacts have already been made by the sub-committee. The Chairman reports that it is a large task but that the work is made much easier because of the contacts made previously by the Chicago Medical Society and the Illinois State Medical Society.

A fourth activity is what we call Pediatric Programs or Post Graduate Courses offered to groups of County Societies and Branch Societies of the Chicago Medical Society. The prime object of this course is to awaken a greater interest on the part of the practitioners of medicine in all matters pertaining to the welfare of the well and sick child to the end that he may be a leader of such activities in his community, *if such activities are necessary.*

The ideal sought for in this program is that the general practitioner who takes care of by far the largest number of children, shall be so well informed that eventually there will be no need of any lay organizations whose object is to look after the welfare of children. The slogan which expresses the ultimate object is *"to make every Doctor's Office a Health Center."*

The committee counselled with the educational department of the Illinois State Medical Society and has formulated its program by and with its approval. The State has been divided into about eight districts with from 8 to 20 counties in each district and the meetings throughout the State have been arranged for by a District Chairman, appointed by the State Committee, for each one of the Districts. This Chairman has been instructed to work through the channels of the regular organized medical societies which consists of proper contact and arrangement with the secretaries of each County Society in the District. These programs are strictly medical in character.

Seven such meetings have been held during July, September and October throughout the State and have been received with a great deal of enthusiasm. These meetings were held at Bloomington, Peoria, Quincy, Monmouth, Rockford, La Salle and Benton. A request came from Rock Island for a program which was cheerfully granted. Invitations have been received for some return engagements next year. We are prepared to present a modified program to the Branch Societies of the Chicago Medical

Society this coming year. Arrangements have already been made for programs to be presented at meetings of the following Branches of the Chicago Medical Society. Irving Park, West Side, North Side, Evanston, Calumet, South Chicago, Southern Cook County. These programs are being voluntarily offered to the Branch Societies and to County Societies free of cost to them for this year. The acceptance is entirely in their hands. We have stressed this point so that there can be no question of a voluntary acceptance of such a program. The committee is strongly of the opinion that, particularly under the present economic conditions, it is utterly impossible for the great mass of practitioners of medicine to go to any medical center and attend a post graduate course; therefore, they have determined to carry this work to the Doctors in the various communities if the Doctors desire to have it. These programs are purely medical. They are altruistic. There is no idea of self advertising; in fact, the men who are giving the programs are giving them at a very definite sacrifice. There are five teams of teachers, each team headed by a teacher of Pediatrics from a medical school or a Children's Hospital. Each one of these teachers picks his group of assistants to present the program. The personnel of each group necessarily will be changed from time to time and for various reasons new men will be asked to present the program.

The following quotation from the address of Dr. Maurice L. Blatt on the subject "Preventative Measures in Childhood" delivered at the Rockford meeting September 20, 1933, gives a clear idea of the importance for practitioners of Medicine to learn and teach preventive medicine as applied to children.

It is difficult to inculcate an appreciation of his part in preventive medicine to a student in a medical college. He is interested in that vast field of diagnosis and treatment of disease, a knowledge necessary before the thought of prevention can enter his mind. There is little in the curriculum to call his attention to prophylaxis. To be sure he learns etiology but in the majority of instances, his past experience is not such that he is able to correlate etiology with the abstract idea of prevention of morbidity and mortality as it applied to him in his capacity as a private physician. His hospital training, the care of sick patients, only accentuates his position in relationship to this problem. This condition is one of the factors which has resulted in the taking over by public health organizations of functions distinctly those of the family doctor and pediatrician. Unfortunately, this department is frequently under the guidance of self-seeking politicians, both medical and non-medical, who because of insufficient study or in their zeal for self-aggrandizement, fail to consider the indirect effect on the public and upon the doctor practicing medicine as a vocation. Brought about by the rapid and extensive increase in medical knowledge which the colleges attempt to disseminate to students and the failure to lay sufficient stress on his duty, there has resulted a usurpation of part of the prophylactic work of the private physician by departments of health. The work had to be done and since the physician did not take the lead himself, the public organizations took up the work. Our patient must be protected and either we have to protect them ourselves or others do it for us. In the opinion of many medical economists, and with this opinion I agree, no organization, no matter how intelligent or how well equipped can take the Doctor's place.

Control of medicine will remain with the physician if he rises to this opportunity; it will be lost and usurped by public bodies if he fails in this trust. A sense of the responsibility for continuing the individuals of the family in good health is of highest importance. To prevent pneumonia in a child, to prevent heart disease complicating rheumatism, the grandmother's chronic nose and throat infections must be treated and her contact with the child interdicted until such time as she is free of infection. This is the Doctor's duty.

We hear much nowadays about the crisis in our Nation, the necessity for emergency actions; the necessity for the Government to borrow and spend huge sums of money; the necessity for the people to exhibit great patriotism in order to preserve our Americanism and American Institutions. So indeed in Medicine; we are at last being awakened to our necessities, to our crises. The practice of Medicine has been so gradually and insidiously encroached upon that only a few were smart enough and far sighted enough to see the trend of medical events. Fortunately, we know the way out. It is through the re-instatement of the family Doctor in his rightful place in Medicine. To aid in the accomplishment of this, the Academy of Pediatrics is dedicated and committed and is attempting to do its part to make all medical practitioners more child conscious, to aid them in so equipping themselves that they will lead and direct all activities for the welfare and the protection of children.

PNEUMOPERITONEUM AND SURGERY IN MANAGEMENT OF ABDOMINAL ADHESIONS*

B. H. ORNDOFF, M. D.
CHICAGO

The fixation of abdominal viscera in such a manner as to be symptom producing is one of the very important problems confronting physicians today in the care of postoperative patients. The fixation of one peritoneal surface to another seems to be a physiologic function rather than a pathologic condition. The capacity of peritoneal surfaces to form fibrinous adhesions with subsequent fixation has made modern surgery of the abdomen possible.

Injuries to the surface of the abdominal viscera are almost always followed by the formation of a fibrinous exudate and in most instances by fibrinous adhesions which tend to limit further trauma, to prevent further distribution of irritating substances and when the usefulness of this change has been accomplished, to undergo dissolution and return normal mobility and the normal intervisceral relations to the abdomen.

It is obvious that if the peritoneum is found not to possess this essential function of forming peritoneal adhesions, whether it be from intrinsic changes causing a loss of this function or from the presence of an agent introduced into the peritoneal cavity which would prevent adhesion formation, abdominal surgery of any character becomes not only very hazardous, but would present entirely new surgical problems that could not possibly be handled by standard methods of operating now in common practice.

While the formation of fibrinous adhesions is an essential function of the peritoneum, the conversion of fibrinous adhesions into organized fibrous adhesions with permanently fixed abdominal viscera cannot be considered physiologic but is usually responsible for pathological changes in the viscera and frequently is symptom producing. The extent of the fibrous adhesions, the degree of restricted mobility, the necessity of motion in the fixed viscus, the character of the function of the affected viscus, the position the viscus is forced to assume, the nervous trauma, circulatory disturbances and other functional interferences together with the mental and psychic influences create a complex which does not constitute a distinct disease entity, but incapacitates the individual to a degree commensurate with the aggregate involvement.

It is probable that every laparotomy is followed by fibrinous peritoneal adhesions, while comparatively few go on to fibrosis. Again, postoperative fibrous adhesions are not frequently sufficiently disturbing to the normal functions of the viscera to greatly incapacitate the individual, but every surgeon who has contributed any considerable share of his work to abdominal surgery has a relatively small, but very definite percentage of patients with postoperative results and complications which require further treatment.

Cases with symptoms due to fixed abdominal viscera range between those with very simple digestive interferences, to cases with symptoms more or less profound in character, simulating at times almost every known abdominal condition and they sometimes include acute intestinal ileus and peritonitis.

Since the very earliest investigations were made leading to modern methods of surgery, ways and means have been devised, experiments have been performed and experiences recorded for the prevention of damaging abdominal adhesions. Investigators have prepared and introduced into animals and into man a long list of substances intended to prevent the formation of undesirable adhesions. In many instances adhesions were prevented to a greater or lesser degree but we must remember that we are striking at the very heart of success in abdominal surgery, for if an agent or procedure would prevent entirely the formation of adhesions, the surgical procedure would in itself become a fatal procedure.

Investigators find that fibrin appears on the surface of the damaged peritoneum within a time period of a few minutes and fibrinous adhesions are well formed within a few hours, while organized fibrous adhesions replace fibrinous adhesions after a period of a few days or several weeks.

The use of air in the peritoneal cavity for the prevention of adhesions was introduced by Bainbridge in 1908. His method consisted essentially of closing the incision around a small rubber tube in such a manner that when air had been

*Read before Section on Radiology, Illinois State Medical Society at Peoria, Illinois, May 17, 1933.

introduced into the peritoneal cavity through the rubber tube, it was quickly withdrawn and the air retained. It was hoped that the air distending the peritoneal cavity would prevent fixation of abdominal viscera to the anterior abdominal wall. The success of this method seems to be very limited and consequently has received but little attention since it was introduced.

My experience with the introduction of gaseous media into the peritoneal cavity began in 1919. The purpose of the work was an endeavor to advance the usefulness of x-ray examination for the diagnosis of pathologic conditions in the abdomen. While the field of abdominal diagnosis was thus being enlarged, the therapeutic use of pneumoperitoneum also received recognition.

The therapeutic introduction of gaseous media into the peritoneal cavity for the release of fixed abdominal viscera was begun early in 1920. The first work was confined to cases of tubercular peritonitis and later cases with postoperative adhesions were treated. In the cases with tubercular peritonitis the results were exceedingly satisfactory inasmuch as the infectious phases of the disease were greatly improved and the visceral fixation was either eliminated or remained without the production of symptoms. In these cases we were also convinced that x-ray treatment into the oxygen filled peritoneal cavity was responsible for a large measure of our success. The x-ray treatment in cases with adhesions, but not tubercular, did not prove to be of benefit and after some experience, it was discontinued. In almost all of this work, the transabdominal route was used for introducing the gas into the peritoneal cavity, even in postoperative pneumoperitoneum it was preferred to the Bainbridge method.

Our clinical experience spreading over the past thirteen years has proven to us that it is a useful therapeutic procedure. During the early part of our work some changes were made in the general management and technique employed, but for the past eight years we have introduced no particular change.

The cases accepted for this work were those who suffered from more or less profound abdominal disturbances and in which the x-ray examinations with pneumoperitoneum warranted the conclusion that the visceral fixation was the principal etiologic factor.

The treatment in brief is as follows: A large diagnostic quantity of oxygen is introduced into the peritoneal cavity and complete x-ray fluorescent screen observations are made and a number of x-ray films are exposed to make permanent records with the patient in all of the positions required in this diagnostic work. Where there is a question of tubercular peritonitis, malignancy, or other conditions, the peritioneoscope is introduced through the abdominal wall and the viscera of the abdomen observed through the lamp and lens system. When the x-ray examinations have been completed, the major portion of the oxygen is removed from the peritoneal cavity and the patient is allowed to carry the remaining portion for from one to four days. The amount of oxygen remaining in the abdomen is then increased by the introduction of about another liter of oxygen. This is allowed to remain for a few days when a larger quantity is introduced and this again repeated, increasing the amount introduced until the patient is carrying quantities of from one to four liters constantly.

The distress noted by the patient following the first pneumoperitoneum is occasionally quite disconcerting, but as the patient becomes more accustomed to the presence of oxygen in the abdomen and the tugging of the viscera while assuming different postures or walking, they manifest very little or no discomfort.

While the patient continues to carry the gaseous media in the abdomen, several things are accomplished:

1. The peritoneum becomes accustomed to the presence of gas.

2. The position of organs which depend upon a vacuum is changed.

3. The disturbances of a physiologic character noted when oxygen is first introduced soon disappears.

4. When the patient is walking, working, or assuming different postures the mobility of the abdominal organs is limited only by their natural ligaments and attachments. Those organs that are fixed pathologically exert a tugging effect upon the adhesions so long as the patient continues to carry the gaseous media in the peritoneal cavity.

5. In many instances the symptoms are alleviated, the nourishing capacity is increased and the feeling of well being is greatly enhanced. In these instances the pneumoperitoneum is con-

tinued at intervals for many weeks and sometimes for many months, but at no time is the peritoneal cavity allowed to become free of the gaseous media.

3. In cases where the wearing of gaseous media in the peritoneum shows very firm visceral fixation, probably of a fibrous character, and so securely fixed that no release is accomplished in the period of two or three weeks, operative intervention should then be instituted. The patient is considered prepared for surgery only when they are thoroughly accustomed to the presence of gaseous media in the peritoneal cavity and when all intestinal reaction has become quiescent.

7. The operation consists of opening the abdomen and releasing the fixed viscera using standard accepted surgical procedures with the least possible intraabdominal trauma, closing the abdomen in the usual way and again introducing gaseous media into the peritoneal cavity and replacing it as rapidly as it is absorbed for a period of about ten days.

8. As soon as the patient is returned from surgery, 1000 cc of tap water is introduced into the colon and this quantity is replenished with 500 cc at intervals of 6 to 12 hours, depending upon the patient's capacity to retain it. The postoperative care includes the desirability of changing the patient's position at frequent intervals from a recumbent to a semi-sitting posture, as well as rotating to both sides, in spite of the distress that must necessarily ensue.

In summarizing the clinical results of cases treated, I feel justified in stating that while many of our cases have not remained symptom free, yet I can say that all of the cases received benefit from the work. Up to date there has been no mortality which could be attributed to the pneumoperitoneum or the surgical interference. Several cases have had repeated operative procedures following the original operation, and we have not been obliged to re-operate in a single case and I am at present keeping informed of more than fifty whose work was done three or more years ago.

Many of the cases have a pronounced keloid tendency. They seem to do especially well. These cases usually have a strong nourishing capacity. There is a class with poor nourishing capacity who seem to have low resistance to infectious processes in the viscera, yet with the "adhesions diathesis." They are more difficult to manage and the end results seem not to be so satisfactory.

In closing, may I reiterate that I realize that the results do not accomplish all that I desire in all of the cases, but in many the results are highly satisfactory, a benefit in almost every case that received the treatment; many are improved satisfactorily with pneumoperitoneum alone, and finally no other treatment seems to offer an equal degree of satisfaction.

2561 North Clark St.

DISCUSSION

Homer D. Junkin (Paris): Mr. Chairman and Members of the Society: It has been my good fortune to have followed Dr. Orndorff for the past twenty-five years. I saw him as a pathologist, as an anatomist, and in surgical experience. I mention this simply because I have a great deal of respect for the radiologist. I find they are men with a big background. They have done much fundamental work to obtain the proficiency they now represent in their field.

In this particular field, those of us who have done general surgery are well aware of the difficulties which we meet in postoperative adhesions. I don't think we have had anything to help us since Crile's message: Sharp dissection, careful peritonization of the raw surface and avoidance of trauma. We have tried various oils intra-abdominally; we have tried various preparations of amniotic fluid which have been put on the market. All of these, to my mind, have failed miserably. Now, Dr. Orndoff gives us a new weapon. In the early part of his work, I remember an article that came out about the dangers of pneumoperitoneum, and there were something like five deaths reported. As I read this article, it told about the gas rising upward, getting about the regions of the neck and the patient finally succumbed after about five or ten hours.

It seems to me that no more skill is required to put a needle into the abdominal cavity than to make a lumbar tap in giving a spinal anesthesia. Perhaps, the latter is the most difficult. Certainly, the deaths attributed to pneumoperitoneum represented a technical error rather than a danger in the procedure.

I have done several cases of pneumoperitoneum for therapeutic purposes. Out West, I encountered a patient who had been in the service where he contracted generalized tuberculosis. Practically all of his organs became involved. First, it was pulmonary. Then the thyroid became infected. Finally, the kidneys became involved as well as the urinary bladder where a large ulcer was found. Then, a series of intestinal obstructions occurred. I operated on him four or five times for this and finally in 1925 I spent about four or five months in Chicago with Dr. Orndoff, and began my pneumoperitoneum work. After I returned home, this patient obstructed very kindly again and I opened the abdomen, removed the adhesions and filled the abdomen with gas. Something like ninety-two pneumoperitoneal

treatments were given over a period of five years. This patient gained in weight from about 115 pounds to 175 pounds. When I left the West and came to Illinois, he moved here and stayed two years. He said this climate was too hot and moved back to Idaho. As far as I know, he is well at this time.

I wish to mention an instance of diagnostic value that came from this work. Out West, I operated on a young lady for appendicitis. A year after the appendectomy, she developed a dermoid of the right abdominal wall at the site of the scar. We referred the case to a Salt Lake Clinic who gave as their opinion: A dermoid of the abdominal wall which involves the cecum. I was perfectly willing to grant that there was a tumor of the abdominal wall but was sure it didn't involve the colon. They had relied entirely upon a simple gastro intestinal examination with the fluoroscope. I did a pneumoperitoneum on this patient. By the lateral position, it was easy to see that the tumor mass was confined to the abdominal wall. The tumor was removed surgically. A year later a small recurrence became evident. The patient had moved to Chicago in the meantime, and Dr. Orndoff irradiated the tumor. It disappeared promptly and has remained well since.

In viewing the apparatus which Dr. Orndoff uses, it seems we have to have a good deal of equipment. This is not essential. One can place the patient on a table in front of the perpendicular fluoroscope and there effectively perform the pneumoperitoneum.

I have one patient who had been operated upon several times for gall bladder disease and postoperative adhesions. About two years ago, we gave a series of pneumoperitoneums without the abdomen being opened. She made a prompt recovery.

On viewing this work, one may fail to grasp the broadness of it. An effective weapon to cope with symptom-producing abdominal adhesions is a great step forward in dealing with these problems of abdominal surgery.

I wish to congratulate Dr. Orndoff upon this paper. It is a masterpiece.

Arthur E. Perley (Quincy): Mr. Chairman, I enjoyed Dr. Orndoff's paper very much. In fact, it is the first time I have heard him speak on the subject of pneumoperitoneum. Wherever I have been and whenever the subject came up, it has always been said that it was a dangerous procedure because there had been some deaths from it. It is apparent that it is not so dangerous if it is properly done. Dr. Santi, in 1927, reported over 1000 cases where it was performed for diagnostic purposes. These cases were done by interns, not more than three being done by a single intern.

Pneumoperitoneum has been done in conjunction with lipiodol. If this is a good procedure, as it seems to be, all I can do is to urge that we try to make it more widespread in different clinics and different parts of the country. I think Dr. Orndoff has shown some reliable results.

As to the subject of adhesions, when I was a student, the professor of surgery used to say we could never tell which patient was going to develop adhesions and which were not. He operated on some cases that had ulcerative conditions; later on they were operated on for some other condition and there would be no adhesions. Others might have the simple chronic type of appendix; but later on they had to be operated on again for adhesions.

In regard to the apparatus, I wonder whether this could not be done with a simple trocar and oxygen tank.

Dr. B. H. Orndoff (closing the discussion): I wish to thank the gentlemen, who have been kind enough to discuss this paper and especially to thank Doctor Junkin for his complimentary remarks.

I did not attempt to discuss special apparatus designed for use in this work. Our work has been entirely an office and hospital procedure. The special table referred to is not necessary, since one can use an ordinary hospital cart and an upright fluorescent screen apparatus very satisfactorily. Doctor Sante has described a very simple and effective method for introducing oxygen into the peritoneal cavity, in which he uses a 50 c.c. syringe with a Murphy drip apparatus, in which cotton has been placed as a filter and with his apparatus, using air to fill the peritoneum, it becomes a very convenient method.

At a recent meeting of the Pan American Medical Association in Dallas, I was very pleased to learn that quite a number were present who were doing pneumoperitoneum in selected cases at this time. As a diagnostic procedure it is not so popular, but as a therapeutic measure, I believe it warrants our attention at this time. It is true that some four deaths were reported from the hands of those who were doing pneumoperitoneum as first introduced, but I have heard of no deaths resulting in the hands of those whose training and experience command their interest sufficiently to warrant their continued interest in this procedure. Our experience permits me to state that with reasonable care it is a safe procedure.

In closing let me say again that this paper has been presented to show the therapeutic phase of pneumoperitoneum and that no attempt has been made to emphasibe the diagnostic possibilities.

MENINGOCOCCUS MENINGITIS; A FURTHER CLINICAL STUDY*

MAXWELL P. BOROVSKY, M. D.
CHICAGO

Attending Pediatrician, Cook County Hospital; Professor of Clinical Pediatrics, Chicago Medical School; Attending Pediatrician, Mt. Sinai Hospital

In a previous report,[1] a series of 190 cases of meningococcus meningitis observed at Cook County Hospital in a period of eighteen months (April, 1927 to September, 1930) was regarded as evidence of an epidemic.

Admissions from 1920 to 1926 numbered from six to sixteen annually and early in 1927 the

*Read before Section on Medicine, Illinois State Medical Society, Peoria, May 17, 1933.

number of cases of meningococcus meningitis increased manyfold. This increase continued through 1930 and the cases to be reported in this article consist of 338 admissions from October, 1928, to June, 1930.

Previous observations that epidemics of meningococcus meningitis occur in cycles of nine to twelve years are not well borne out by this report, for the Chicago district was in the throes of an epidemic from 1927 to 1931 with only brief and slightly restricted intervals.

The epidemiologic fact that meningococcus meningitis is only mildly contagious is again sustained by the fact that among these 338 there were only eleven instances of more than one case in a family. This checks well with Neal's[2] observation of 13 multiple cases among 500 and my previous figure of five among 190 cases.

The 338 cases were admitted to the Cook County Hospital on the services of Drs. Hoyne, Bower, and Borovsky from October 1928 to June, 1930. Of the 338 cases, 239 or 70.7% occurred among white patients and 88 or 26% among negroes.

The age of these patients varied from three months to eighty years, and 177 (53.3%) were under 14 with 161 (47.7%) above 14. The mortality among this group was considerably higher than in the previous series. The general death rate was 57.1%. However 63 of these patients died during the first 24 hours after admission to the hospital. They constitute then, 32.6% of the total deaths. It is obvious that these patients entered our institution in a very serious, if not moribund, condition and had little opportunity to show any response to therapy. It is not without practical medical thought to exclude these from the general mortality list and to include only those cases that "had a chance." This corrected figure would then be 47.2%. Fifty-two deaths occurred among the 88 negro patients or 59.1%.

The mortality was highest under two and over forty years of age. No patient over fifty recovered from meningococcus meningitis, a total of twenty-five deaths. The lowest mortality occurred from four to five and from six to eight years.

1. A clinical study of Meningococcus Meningitis. Amer. Jour. Med. Sci. January, 1930. No. 1, Vol. CLXXLX, P. 82.
2. Abt's Pediatrics, Vol. VI, P. 423.

TABLE 1. MORTALITY ACCORDING TO AGE

Age in years	No. of cases	Recovered	Died	Mortality Per Cent.
Under 1	12	4	8	66.6
1 to 2	7	2	5	71
2 to 3	28	14	14	50
3 to 4	10	3	7	70
4 to 5	25	18	7	28
5 to 6	10	4	6	60
6 to 7	11	6	5	45
7 to 8	13	11	2	15
8 to 9	13	11	2	15
9 to 10	12	6	6	50
10 to 11	15	10	5	33
11 to 12	5	3	2	40
12 to 13	9	6	3	33
13 to 14	7	4	3	43
14 to 20	37	18	19	51
20 to 30	50	11	39	78
30 to 40	30	9	22	70
40 to 50	25	3	22	88
50 to 60	9	0	9	100
60 to 80	8	0	8	100

TABLE 2. MORTALITY WITH REFERENCE TO SYMPTOMS AND COMPLICATIONS

	Total Cases	Recovered	Died	Mortality Per Cent.
Coma	74	18	56	78
Semi-Coma	53	28	25	47
Stupor				
Drowsiness				
Delirium	49	18	31	63
Convulsions	29	12	17	58
Petechiae	62	26	36	58
Chills	27	8	19	70
Herpes	10	5	5	50
Strabismus	25	13	12	48
Ptosis	1	0	1	100
Panophthalmitis	2	0	2	100
Iridocyclitis	1	1	0	0
Nystagmus	11	7	4	36
Arthritis	1	0	1	100
Deafness	21	19	2	9
Supp. Otitis	1	1	0	0
Neg. Brudzinski	11	3	8	73
Neg. Kernig	12	3	9	75
Extreme				
Opisthotonous	36	29	7	19
Slight Retraction	219	94	125	57
Bulging Fontanelle	6	1	5	83
Normal Fontanelle	2	1	1	50
Previous Trauma to Head	9	4	5	55
Multiple Cases in One Family	11	8	3	27
Hemorrhagic Nephritis	1	1	0	0
Abscess at Sight of Puncture	2	0	2	100

Again, as in the previous report, the patients brought to the hospital in coma showed a high mortality, namely 78%, as compared with those designated as semi-comatose, stuporous, or drowsy, whose mortality was 47%, and the delirious patients who showed a mortality of 63%.

Convulsions occurred in 29 patients and the death rate among this group was about the same as the general mortality, 58%.

In 32 patients, 18.3% of the total, petechiae

were present. This was usually a general skin manifestation with lesions on the chest, abdomen, and extremities, but occasionally very limited in their distribution. It is extremely interesting to note that the death rate in this group is the same as the general mortality in contradistinction to the prevalent idea that these cases offer a much more serious prognosis than cases without a hemorrhagic tendency. In the previous series, among 28 cases with petechiae there was a mortality of 64.2%.

The gravity of the occurrence of chills as a symptom in cases of meningococcus meningitis is also to be noted. Patients with this part of the picture died in 70% of the cases.

Of unusual interest, and what seems paradoxical to everyone who is at first confronted with this information, is the fact that the more severe the opisthotonous the better the prognosis. It has been a very common observation that patients with arching of the back and retraction of the head resembling the arch of a rainbow usually were lucid during their illness, responded exceedingly well to therapy, and most commonly recovered. Of 36 patients described as in extreme oposthotonous, 29 or 81% recovered. On the other hand, 219 patients reported as showing slight retraction of the head showed a mortality of 57%.

In our regular rounds in the meningitis wards it was interesting to see how often the supposition, "the more extreme the opisthotonous the better the prognosis" was true, and supervising nurses and internes soon took hold of this observation and verified this statement themselves.

These patients are usually clear mentally because of the greater limitation of the exudate to the base of the brain and the relative or complete freedom of the cerebral hemispheres from pus. The degree of opisthotonous is in a measure proportionate to the basilar involvement.

Negative Brudzinski and Kernig signs were observed in 11 and 12 cases respectively, and usually in the more severe cases as evidenced by the mortality of 75%. This is clarified still further by the presence in this series of twelve patients under one year of age where the mortality is high and it is a well known fact that definite meningitic involvement may be present without either the Kernig or Brudzinski tests being positive. Only one-half of these negative cases occurred in patients over one year.

Bulging fontanelle was observed in six infants and five of these died, a mortality of 83%. Normal fontanelle was present in two infants only one of whom died.

Previous trauma to the head or spine within ten days prior to the onset of epidemic meningitis was discovered in nine instances, five of whom died, a 55% mortality.

Of the eleven multiple cases only three, or 27% died. This was undoubtedly due to the fact that medical advice was sought early, because of the previous and usually very recent experience.

Herpes was present in only ten patients and was equally distributed in fatal and recovered cases.

Strabismus was observed in 25 patients, 13 of whom recovered, a mortality of 48%. This was a definitely transitory complication for in each case in which this complication occurred, a reexamination six months after discharge from the hospital revealed the return of normal function of the extrinsic eye muscles.

Deafness was discovered in 21 patients, 19 of whom recovered, leaving a very low mortality of 9%. This deafness was usually bilateral and only occasionally unilateral. The patient's hearing was completely lost due to nerve destruction and in practically all cases was permanent.

The number of cases of deafness would undoubtedly have been higher if many of the several cases that died earlier in their stay in the hospital had survived. Many of these patients remained in coma during their brief hospital residence and hearing tests were impossible.

Suppurative otitis media was extremely rare. It occurred in only one patient and that patient recovered.

TABLE 3. MORTALITY ACCORDING TO DAY OF ILLNESS ON ADMISSION

Day of Illness	No. of cases	Recovered	Died	Mortality Per Cent.
1	42	17	25	59
2	84	37	47	56
3	59	30	29	49
4	37	14	23	62
5	25	15	10	40
6	4	3	1	25
7	11	4	7	63
8	1	0	1	100
9	1	0	1	100
10	5	3	2	40
12	1	0	1	100
15	1	0	1	100
20	1	1	0	0

From a study of table 3 it is quite apparent that the day of illness when treatment is begun is not as important a factor as might be imagined for a high mortality occurs in those patients who enter the hospital during the first two days of their illness. The mortality is considerably lower among those patients who have their specific treatment begun on the third, fifth or sixth day of the disease. This statement is contrary to all the laws of therapeutics but is probably explainable by the fact that patients who are sick enough to seek medical aid early in the course of their illness are the ones with the severest types of infection. They therefore have a poorer chance of recovery than those who show fewer manifestations of the disease and remain at home either under the care of relatives or unsuspecting medical men who are treating the patient for a "stomach upset" or the grippe. The patients in the latter group have a milder type of infection and even though treatment is started later in the course of their illness they have a good chance for recovery. In fact, some of these patients may recover without any specific treatment.

The average stay in the hospital of recovered cases was 23 days.

TABLE 4. STAY IN THE HOSPITAL OF RECOVERED CASES

No. of Days	No. of Patients	No. of Days	No. of Patients
8	1	27	7
9	2	28	6
10	2	29	2
11	3	30	9
12	6	31	1
13	6	32	3
14	5	33	1
15	10	34	2
16	8	35	7
17	4	36	1
18	6	38	3
19	7	39	1
20	2	40	2
21	4	41	2
22	3	43	2
23	9	44	1
24	4	45	1
25	5	46	1
26	6		

The shortest period in which a patient was discharged as cured was eight days, and the longest period was forty-six days. The greatest number of patients, however, left the hospital on the fifteenth and sixteenth days which may be considered a fair average for uncomplicated cases.

It is a well known fact that the meningococcus is less abundant in the spinal fluid of a case of epidemic meningitis than in a parallel case of suppurative meningitis caused by the streptococcus, staphylococcus, or pneumococcus, but if a careful search is made in a smear of the fluid obtained at first spinal puncture, i. e. before serum administration, the meningococcus should be discovered in about 90% of the cases. Culture of the spinal fluid soon after removal, or still better, collection of the fluid directly from the needle onto the culture medium will bring this percentage up much higher.

The average cell count in the cerebrospinal fluid on admission was 10,546. The lowest count was 600, and the highest 74,000. There were only six counts under a thousand, and four over 50,000. The differential cell count in every instance was recorded as polymorphonucleosis ranging from 75 to 95%.

In almost every case the spinal fluid was cloudy on first examination varying in degree from faint cloudiness to a thickly purulent material that scarcely ran through the needle. Occasionally the fluid was barely detectable as cloudy with a cell count of 600 or 700, but after the serum administration and further development of the meningitic picture, the fluid became definitely cloudy and the cell count mounted to the thousands. The globulin tests were universally positive.

The blood showed a leucocytosis in every case in which it was examined, the average being 18,070. The lowest white count was 9,600 and the highest 60,000.

Antimeningococcus serum of the polyvalent variety was used in the treatment of these cases. No difference was found in the therapeutic value of any of the brands employed. The occasional run of satisfactory or unsatisfactory results was attributed more to the virulence of the epidemic organism at a given time than to the efficacy or inadequacy of the serum used. Occasionally, improvement was not apparent until a change was made from one brand to another, but this was considered accidental.

A truism may be applied to the therapeutics of cases of meningococcus meningitis, namely; "if you don't get drainage you can't get recovery." This is well substantiated by our results and caused us to immediately resort to cisterna punctures when drainage by spinal punctures was becoming inadequate. The amount of drainage

necessary varies with the age of the patient and is roughly 15 to 25 c.c. from one to five years of age grading up to 40 or 50 c.c. in adults. This is again proportionate with the amount of serum administered intra-thecally, namely 15 c.c. to children under five and averaging up to 30 c.c. in older children and adults.

The average amount of serum used in recovered cases was 188 c.c. intra-thecally. The smallest amount in a case that recovered was 30 c.c. intraspinally in two administrations. The largest amount used was 645 c.c. In ten instances 60 c.c. or less was sufficient to effect a cure.

The routine of daily intra-thecal injections of serum was carried out, the administrations usually being 5 to 15 c.c. less than the amount of fluid withdrawn. This was continued until the fluid became clear with a diminution in the cell count to a few hundred. Daily spinal puncture drainage was continued, however, until the pressure returned to normal and the cell count returned to 100 or less.

If this routine is followed less likelihood of recurrences is present. The idea that serum administration may be responsible for a continued cell count in the thousands is not upheld. Any cell count of that degree is definitely indicative of the continued presence of active infection and treatment should be continued. The irritative reaction of excessive serum may be responsible for a cell count of a few hundred but hardly of a few thousand.

Too early discontinuance of serum therapy is a grave error. In summary, intra-thecal serum injections should be continued until the spinal fluid becomes clear and the cell count is reduced to 100 or less with the subsidence of meningitic symptoms and the absence of temperature.

If fever and meningitic symptoms persist in spite of clear spinal fluid, daily spinal drainage should be resorted to until the patient is clinically well. Any clouding of the spinal fluid or substantial increase in the cell count calls for re-institution of intra-thecal serum therapy.

The average number of spinal punctures was 8.9 in recovered cases. The smallest number was one, and the greatest twenty-nine. Out of 146 recovered patients, sixteen required three or fewer spinal punctures.

Cisterna punctures have a definite place in the therapy of meningococcus meningitis. Our routine was to perform spinal punctures as long as adequate drainage was possible by this means and the performance of cisterna puncture when sufficient fluid for one reason or another, was not obtainable by the lumbar route. This procedure was extremely practical in cases of marked opisthotonous where sufficient arching of the back was not possible for the performance of a lumbar puncture.

Total cisterna punctures on recovered patients was 279 which were performed on forty-eight individuals, an average of 5.8% per patient.

Total cisterna punctures on fatal cases was 126 performed on 38 patients, an average of 3.3 per patient. Ten more patients recovered after cisterna punctures than died.

Intravenous antimeningococcus serum was administered to forty patients in one or two 15 c.c. injections, the first on admission, and the second injection one to fourteen days later. The mortality in this group was 65%.

The efficacy of this form of therapy must again be questioned for in this series as in the previous one the same relative increase in mortality occurred, namely eight per cent. over the general mortality.

The patients that received intravenous therapy were chosen at random, i.e. not only the extremely sick patients received the intravenous serum.

The therapeutic value and the rationale of this treatment is very much doubted even in cases seen early that have an unquestionable meningococcemia. The presence of the meningococci in the blood is usually short for their choice of tissue habitat is the meninges. When they have implanted themselves in the meninges intrathecal therapy is the one of choice. Patients with petechiae are very likely to have a positive blood culture, but they respond very well to intraspinal therapy alone. Sudden deaths and severe anaphylactic reactions nearly fatal have been noted too frequently to allow the recommendation of intravenous therapy to go unchallenged. This is more likely to bring about unfavorable results if the intravenous injection is given a few days after intrathecal therapy was begun. Three patients in this group had violent reactions following intravenous injections. A description of one as given by the interne follows: "The patient was given 20 c.c. anti-meningococcus serum in-

travenously. The warmed solution was given slowly but was followed by marked shock and collapse with apparent respiratory paralysis. Breathing verged on the Cheyne-Stokes type and the face became markedly cyanotic. The treatment consisted of external heat, reverse Fowler's position, adrenalin mins. 5 intravenously, and mins. 10 subcutaneously. Atropine grain 1/100 and camphor 1 c.c. hypo at fifteen minute intervals. Then adrenalin mins. 15 every two hours for six doses. The patient finally recovered."

If there were clinical evidence of the value of intravenous therapy in meningococcus meningitis such arguments would not be decisive but our figures show an increase rather than a decrease of mortality in the cases that receive intravenous anti-meningococcus serum in addition to intraspinal injections.

In this series there were fourteen recurrences of the meningitic picture with secondary clouding of the spinal fluid. There were two cases on the 11th day, two on the 13th day, two on the 15th day, one on the 19th, two on the 21st, one on the 22nd, two on the 23rd, one on the 31st, and one on the 50th day after the initial onset of the disease.

Eight of these patients subsequently died, a mortality of 57%.

RECURRENCES

Day	Secondary cell count	Recovered	Day of Recovery	Died	Day of Death
11	32,000	0	..	1	38
11	.....	..	..	1	25
13	2,925	1	45	..	..
13	.....	1	35	..	..
15	7,200	..	..	1	..
15	.....	1	38	..	..
19	.....	..	..	1	26
21	4,200	1	43	..	..
21	.....	..	..	1	32
22	10,000	1	27	..	..
23	.....	..	..	1	..
23	.....	..	..	1	31
31	.....	1	50	..	..
50	.....	..	..	1	..

Recurrences will be detected much earlier if spinal puncture is resorted to immediately if there be a secondary rise of temperature and return of rigidity of the neck, Kernig and Brudzinski signs. A discovery of reclouding of the spinal fluid and increase in cell count calls for another intensive course of intrathecal therapy until the spinal fluid is clear again.

Sixty of the recovered patients were re-examined two to fourteen months after discharge from the hospital. Of sixty patients, 37, (53.3%) were entirely free from complaints and two patients described their general well-being as better than before the advent of the attack of meningitis. In my previous series 55.7% were symptom free.

Deafness was the outstanding sequela, which occurred in 14 cases, (23.3%). In the previous series this figure was 25.4%. Five of these cases were unilateral, and nine bilateral. Strangely enough, as reported before, there were again three patients who developed their deafness three months after discharge from the hospital. These were all bilateral cases whose hearing had been perfect when they left the hospital.

Satisfactory explanation of this strange observation is not at hand, but theoretically it could be due to degeneration of the eighth nerve caused by scar tissue contraction at the point of passage through the meninges.

The deafness following meningococcic meningitis is usually a nerve deafness, and with this nerve destruction hope for the recovery of hearing is despaired of.

Six patients complained of occasional headaches but none of these were ever severe enough to interfere with daily activities and some may have been on a psychic basis.

No instance of permanent strabismus was discovered in this follow-up series although there were thirteen recovered patients who developed strabismus. As in the previous series this proved to be a temporary complication.

Five patients had gained weight within a few months after discharge from the hospital, and one had lost weight. Only one complained of poor appetite. Five were slightly irritable since the attack of meningitis. One child was mentally dull since the illness, and one showed an improved mentality. Two told of occasional attacks of dizziness. Three complained of occasional lumbar pain.

None of these complaints were severe except those referable to deafness.

SUMMARY

Three hundred thirty-eight consecutive cases of meningococcus meningitis are reported with reference to mortality according to age, color, day of illness on admission, clinical picture, type of

treatment, the prognostic significance of certain symptoms and complications, laboratory findings, and the reports of re-examinations made months after the patient's discharge from the hospital.

The great similarity in the observations made on this group of patients and those in the previous series is to be noted. Special emphasis is made on the observation that the patients with extreme opisthotonus seemed to have a better chance for recovery than those with only slight retraction.

The apparent futility and the accompanying dangers of intravenous anti-meningococcus serum therapy is again noted.

310 S. Michigan Avenue.

DISCUSSION

Dr. Gerald Cline, Bloomington: It seems as we all plow through the realms of medical practice, we through time, learn to accept and base our opinions a great deal upon statistics. Dr. Borovsky has given us an unusual paper in that he is able to report over three hundred cases of meningitis, which is a great many more cases than the average man will see in a life time.

I was interested to know that his cases showing petechiae gave a bad prognosis, and that those with more pronounced signs of opisthotonus gave a better prognosis. Certainly these two findings are valuable to us all. His statement that the use of different sera seemingly did not show any advantages in the management of his cases also agrees with my meager experience.

We have all thought and been taught that seldom does more than one case appear in the same family. I am sure this is comforting to us as physicians.

We men in the smaller communities have quite a different problem to face in the diagnosis and treatment of such cases. We are not equipped with contagious hospitals. Everyone in the community usually knows the patient; has a fear of the disease, and naturally if the patient is on the floor of the hospital with other cases, no matter how well isolated, he is as foreign as a case of leprosy and is a detriment to the hospital financially. Therefore, most of our cases have to be managed at home, where it is nothing to do a spinal puncture by flash or lamp light and with no competent help. The fluid then has to be brought into town to the hospital for analysis. Most hospitals can not afford, because of the infrequency of the work, to be equipped for cultural laboratory methods.

I agree with frequent multiple drainages and administration of serum and particularly believe it is very important not to stop the drainages too soon. Again as stated before we lack laboratory facilities and, therefore, I feel better if I drain them a few times more than necessary rather than not enough. Also, I feel complications are less in these cases.

Serum reactions which Dr. Borovsky did not mention, but I am sure he encounters, are sometimes quite a disturbance to us in small community practice where we do not have internes and nurses to help us with our cases.

I want to mention one case I had of chronic or recurrent meningococcus meningitis. The child was sick a year before with what sounded to me like mild symptoms of cerebral irritation; with headache, vomiting, backache, etc. No diagnostic spinal drainage was done and with ordinary symptomatic treatment he apparently made a good recovery. When he came to see me he again had mild symptoms comparable to the history of those of last year. My diagnostic fluid showed a high cell count and meningococci (gram negative) on direct smear. I immediately started serum treatment which I feel sure was responsible for an unexpected death, preceded by signs and symptoms of an actute flare up. Another specimen of fluid was sent to a Chicago laboratory and report returned negative for meningococci. Finding myself in quite a difficult situation with the parents, fluid was sent to another laboratory which agreed with my original diagnosis. Postmortem finding revealed an old chronic inflammatory process of the meninges with adhesions. I relate this case to show how questionable laboratory reports can make life very uncomfortable to you as the physician. I suppose my treatment and removal of fluid released some old adhesions, causing an acute flare up.

Again, I enjoyed this unusual paper and congratulate the author. He has brought us a valuable and timely paper.

Dr. W. L. Crawford, Rockford: I want to discuss one point that Dr. Borovosky brought out about getting spinal fluid with a negative report on meningococci and then having the patient develop definite positive spinal fluid symptoms. In connection with his statement about petechiae and pneumococci, I remember the remarks made by Stafford McLane a couple of years ago when he gave a number of case reports where he had done an early spinal puncture and had normal cells and a negative culture. During the period of meningococcemia, where the germs were circulating for several days, in a series of from 25 to 50 cases where they had taken blood cultures from the petechiae, they got better than 50 per cent. positive cultures from these spots during the period when spinal fluid was negative. This corresponds with the high percentage of blood culture that Dr. Borovsky brought out. Keeping this in mind, then, you can make a positive diagnosis in a number of these cases at a time when it would do the patient a great deal of good and before there is a lot of meningeal involvement.

Dr. M. L. Blatt, Chicago: The subject of epidemic meningitis is one of sufficiently serious import to be worthy of additional discussion. Since the epidemic reported from Indianapolis, three years ago, the middle west has seen a larger number of cases than were reported during a number of preceding years. This unfortunately is continuing at this time. It is important that every physician recognize that we are in a period

of high incidence of epidemic meningitis. Knowing this, he is on the look-out for disease and picks up some of the cases which might otherwise be undiagnosed or diagnosed late.

I want to emphasize that even with the most typical cases the first spinal puncture may be absolutely negative. I have seen three such cases. One clinically typical case went on to death because the diagnosis was not made by the first physician to make the first spinal puncture. The fluid was clear. The case was not treated and 24 hours later when the spinal fluid was crowded with meningococci and pus cells, serum proved unavailing. The presence of a large number of pus cells is sufficient indication for a probable diagnosis and the use of antimeningococcic serum is indicated. The spinal fluid may be clear although the cultures frequently are reported as positive for meningococci after 24 hours incubation. As regards treatment, I have recently seen two children come into the hospital with no cerebral symptoms whatsoever. The patients seemed and looked well except for high temperatures and multiple petechiae. These were typical of meningitis in the absence of evidence of endocarditis. Both these patients were treated intravenously with serum. Within 24 hours, one child was entirely well and wanted to go home; the other was apparently entirely well in three days. Intravenous therapy is indicated in such cases as these. Its immediate use is life saving.

Dr. M. P. Borovsky, Chicago (in closing): I want to thank the discussants very much for their interest in this paper.

We do see a large number of serum reactions just as with any foreign protein injection and some of these reactions are quite severe. Some patients develop arthritis and adenitis but they respond in one to three days to adrenalin and ephedrine.

The case that Dr. Cline mentioned of chronic meningitis might have been a recurrence. I had one recurrence as late as seven months after the patient had recovered. It is perfectly possible that there would be a small pocket of infection that is walled off which breaks open and sets up a new infection.

Dr. Blatt's point was very much in order. The early clear fluid is not uncommon. After the first spinal puncture there may be an influx of meningococci, the real spinal fluid picture is set up and the diagnosis can be made very quickly. Surely intravenous therapy is definitely indicated in meningococcemia as Dr. Blatt mentioned. It is only in cases where we have a full-blown typical meningitis that my feeling is that the severe reactions produced are not worth the effect produced by intravenous treatment. I feel that the intravenous therapy, so far as we know, does not do a great deal of good for meningitis. There has been much controversy over that point but when you see patients go into anaphylactic shock from intravenous therapy and when we see cases with petechiae do well on intraspinal therapy alone, we begin to question the value of intravenous treatment along with its possible dangers.

DIABETES MELLITUS AND ESSENTIAL HYPERTENSION.

A Theory as to Their Etiology and Treatment.

James H. Hutton, M. D.,
Chicago.

Our present theories of diabetes mellitus and essential hypertension are not adequate. The pancreatic deficiency hypothesis of diabetes is not supported by clinical and pathological evidence. In hypertension we are practically without a hypothesis. Our purpose here is to present a theory of pituitary and adrenal overactivity as the cause of most cases of diabetes and essential hypertension. It is quite likely that except for Banting's life-saving discovery of insulin the pancreatic theory of diabetes would have been discredited long since by the pathologists.

It is our idea that carbohydrate metabolism is controlled by a balanced mechanism consisting of the pancreas, the pituitary and the adrenals. Hitherto we have not recognized the pituitary and adrenal factors, but we have looked on diabetes mellitus as the result of too little of the pancreatic contribution — hypoinsulinism. Our belief is that it usually results from *too much* of the balancing factors, the pituitary and adrenal secretions, and that the more logical mode of attack is by attempting the regulation or reduction of the secretion of the pituitary and adrenals, the pituitary probably being the primary offender. The fact that insulin relieves the symptoms of diabetes is no argument against the validity of this idea. We are in total ignorance as to how insulin acts and it might as logically be assumed to have a counteractive effect on the pituitary and adrenal secretions. This has already been suggested indirectly by Cushing.[1a] Furthermore, the fact that Cushing[2a] relieved a severe and intractable case of diabetes with the x-ray is another bit of evidence in favor of this theory. Our own experience supports this idea.

The method of treatment here suggested offers possibility of control, if not absolute cure, of hypertension, a syndrome, for which up to this time, we could offer not the slightest hope. For diabetics it offers the possibility of an eventual

cure. Even if it does no better than to control it, as insulin does, the patient would still be better off because he would be free from the inconvenience and danger of constant hypodermic medication.

DIABETES

Evidence in favor of the Pancreatic Theory. Removal of the pancreas in experimental animals causes diabetes—post hoc propter hoc reasoning.

Insulin relieves most of the symptoms in the human as well as the experimental animal.

The pancreas is said to be the seat of pathologic changes in the islet tissues.

All other evidence is secondary to these three main pillars.

Evidence against the Pancreatic Theory. There is no distinctive pathological lesion present in the pancreas in the vast majority of cases. It is reported from Departments of Pathology that only rarely is any pathology found in the pancreas.

Anderson[3] reports the case of a man, aged 33, who suffered from diabetes for fourteen years and at the age of 33 died from coronary disease with marked arteriosclerosis. The pancreas of the patient appeared normal.

Epstein[4] says that in patients dying of diabetes mellitus, insulin could be discovered not only in the pancreas but elsewhere in the body and that fatal diabetes mellitus may exist, and yet insulin be present in the body in quantities that should be considered sufficient to prevent hyperglycemia.

Boyd[5] says: "When a person dies of diabetic coma one might expect to find the same profound pathological change in the pancreas as is seen in the kidneys in uremic coma. Such is not the case. To the naked eye the pancreas usually appears quite normal. . . . I have never encountered a specimen showing any gross change which may not be seen in the non-diabetic. On the other hand, the pancreas may be almost entirely replaced by tumor growth without the occurrence of any symptoms of diabetes.

"The microscopic appearance is also disappointing, although here more definite changes are encountered. A survey of the literature will reveal a great variety of lesions. The fibrosis of chronic pancreatitis may be so extensive that the islets as well as the acinar tissue are destroyed. But diabetes is seldom associated with chronic pancreatitis.

"It is worthy of note that in the notoriously severe diabetes of childhood neither fibrosis nor hyalinization are observed in the islets."

Wullenweber[6] points out that whenever diabetic symptoms are detected, changes in the pancreas are generally thought of, although it is known that carcinoma and necrosis of the pancreas may exist without causing diabetic disturbances and, on the other hand, glycosuria is often observed in disorders of other organs. He reports one patient with severe diabetes in whom at postmortem bilateral tumors in the suprarenal medulla were found. Another had a large hypernephroma. The pancreas did not show pathologic changes.

Joslin says: "Indeed, one is tempted to wonder whether the changes in the islands may not be the result rather than the cause of diabetes."[1b]

"One-tenth of the pancreas may, but usually does not, suffice to prevent diabetes. Whatever the process or processes may be which cause diabetes it is justifiable to infer from these experiments of Allen that at least 90 per cent. of the gland's efficiency must be impaired. . . . No single distinctive lesion of the islands was encountered in 26 of my cases reported in the Warren and Root series or in 10 cases of diabetes in children studied by Warren. * * * This was true even of the young uncomplicated cases of diabetes among whom, if among any group, one might expect uniformity of pathological appearance if there were one definite causal agent giving rise to the disease."[1c]

"We have found no pancreas in this series, no matter how severe the disease process or how marked the changes in the islands, in which a greater or less number of apparently normal islands could not be found. . . . The acinar sclerosis was not apparent in any case much under two years' duration even though the islands showed considerable change."[1d]

"Warfield makes the statement that to his knowledge 'no case of disease of the head of the pancreas has ever been followed by diabetes.' "[1e]

It is unreasonable to believe that any organ could be so badly diseased as to be responsible for the patient's death without some evident pathology being found at postmortem. This is particularly true of the pancreas of which we are told 90 per cent must be removed in order to produce diabetes in the experimental animal. Curiously, we have tried for forty years to compress our clinical and pathological observations into our conception of the etiology of diabetes. Our pathologists have encouraged us in this futile effort by neglecting to examine the adrenals and the pituitary, two structures that have long been known to be influential in carbohydrate metabolism.

Clinical Evidence. The deficiency idea does not square with common sense or experience.

Hyperparathyroidism is characterized by hypercalcemia, increased excretion of calcium in the urine and by a reduction of calcium in the bones. Diabetes is an almost exact counterpart except that sugar is involved in one and calcium in the other.

The syndrome of hyperinsulinism, first described by Seale Harris,[7] has been due in only a few cases to tumors of the pancreas. In most instances the pancreas has been normal in gross and microscopic appearance. In one case reported by Winans[8] the pancreas was only one-third normal size. Microscopically its tissues appeared to be normal. Furthermore, in this case injections of pituitrin relieved the symptoms better and for a longer time than anything else. In others mentioned by Harris[9] suprarenal gland by mouth relieved the symptoms. Descriptions of some of the cases reported sound like clinical cases of hypopituitarism. Winan's experience in relieving the symptoms by injections of pituitrin strengthens the suspicion that at least some of these owed their difficulty to pituitary deficiency. Hypophysectomized dogs experience hypoglycemic crises like cases of "hyperinsulinism.' [20]

Evidence in Support of the Pituitary-Adrenal Theory. Clinical Evidence. Glucose Tolerance Curve. The present theory is that glucose stimulates the pancreas to increased production of insulin resulting in a decline in the curve to a point below the fasting level. It is just as logical to assume that the pituitary takes care of this load of sugar as rapidly as possible but that the increased exertion causes some exhaustion with the result that the blood sugar falls below the starting point.

Cases of hypopituitarism involving the growth hormone of the anterior lobe frequently show sugar curves that rise slightly or not at all above the fasting level and decline steadily to below 60 mg. at the end of two hours. Cushing[10] first called attention to the increased sugar tolerance of animals having hypopituitarism and clinicians have ever since noted this fact in their clinical cases. Simmonds' disease is characterized by low blood sugar and high glucose tolerance. Gigantism is sometimes accompanied by diabetes.

Rowntree[11] qoutes Cushing and Davidoff. "They believe that there is ample support for the belief that the melliturias which occur in about 25% of all cases of acromegaly may be similarly ascribed primarily to the hypophysis. * * * They conclude that the glycosurias accompanying acromegaly are ascribable primarily to the hyperpituitarism. . . . Cushing and his associates believe that the increased rate" (BMR in exophthalmic goiter) "and diabetes mellitus are both due to diseases of the pituitary and can both be adequately treated by partial extirpation of the hypophysis. . . . Borchardt maintains that in 35% of cases of acromegaly, glycosuria is evident, that it is an early manifestation and that later in the disease hypopituitarism replaces hyperpituitarism and that glycosuria disappears spontaneously."

F. N. Allan[11] says: "The incidence of diabetes with hyperpituitarism is certainly too high for such chance relationship. . . . The chief theories which have been proposed in this connection are that excess pituitary secretion may neutralize the effect of insulin or by stimulation of the pancreas lead to its exhaustion."

Fenn and DeTakats[12] noted improvement in carbohydrate tolerance following section of the splanchnic nerve.[5]

Adrenal tumors are many times associated with diabetes.

Worry, fear, anger, excitement, the same factors which stimulate the adrenals, aggravate diabetes, increase blood sugar, sometimes to the point of glycosuria in presumably normal persons, and raise blood pressure.

Female diabetics with ovarian insufficiency have an improved carbohydrate tolerance when their ovarian insufficiency is corrected.

Joslin[1a] quotes Cushing as saying that "insulin and posterior-lobe extracts have been shown to be, and insulin and anterior-lobe extracts may be presumed to be, counteractive in their effects."

A good many diabetics show evidence of an excess of the growth hormone and a shortage of the sex hormone, that is, they are eunuchoid types of individuals.

Cushing's[2a] cases of pituitary basophilism showed glycosuria and hyperglycemia and at least one case had a severe diabetes which could not be controlled by insulin and diet. This particular case was relieved by doses of x-ray to the pituitary.

Joslin says:[1f] "Thirst with a history of being 'a water drinker as long as I can remember' is a symptom not uncommonly volunteered by diabetic patients. This symptom together with the known tendency of diabetic children to be above height for their age at the onset of diabetes, their acknowledged mental procociousness, the proved development of their bones a year in advance of their age, the peak incidence of diabetes in

children being at maturity, * * * the occurrence of glycosuria in pregnancy, the general frequency of obesity prior to the onset of diabetes in adults, all suggest that the pituitary is concerned in the development of diabetes."

Martin[13] says that it is plausible to assume that diabetes and acromegaly are related through a hyperfunction of the pituitary with coincident alterations in the pancreas.

Priscilla White[14] says: "The excess growth of our prediabetic patients may well be a phase of pituitary activity. Whatever the explanation may prove to be, it is significant that preceding the onset of diabetes in the child it is the rule to find for the age an excess of height."

Moehlig and Ainslee[15] say: "The pituitary gland, regulating the suprarenal cortex and other mesodermal tissues, plays an important role in the pathogenesis of diseases associated with abnormalities of cholesterol metabolism. Such diverse clinical entities as atherosclerosis, hypertension, certain types of nephritis, disturbances in the volume of blood, cholelithiasis, diabetes and uterine fibroids are found to have important relations to states of pituitary hyperfunction and exhaustion."

Moehlig and Bates[16] record their impression that eosinophilic and basophilic adenoma have some features in common. Hypertension and hyperglycemia, as well as hypertrichosis and polycythemia, are common in both.

Evidence from the Pathologists. Moehlig and Bates[16] say:

"Kraus and Traube examined two hundred and thirty-two pituitaries of persons who had been ill but a short time, so that no change in the pituitary would be expected from the illness. Another group was obtained from persons who had either died by accident or had committed suicide." Among the conclusions were the following:

"Normal persons of hypersthenic habitus show, as a rule, a marked increase of the basophil cells as compared with those of the healthy mesosthenic habitus.

"Persons with diseases which attack, as a rule, the hypersthenic type, such as essential hypertension, vascular sclerosis, contracted kidneys, constitutional obesity and, in a certain degree, chronic alcoholism, progressive paralysis and aortitis of syphilitic origin, show a very high percentage of basophil cells and a marked increase of the basophil cells as compared with the mesosthenic type.

"Persons with high blood pressure, chronic nephritis and so-called secondary contracted kidneys also have a marked increase of the basophil cells as compared with the normal mesosthenic type.

"Asthenic persons, such as the diabetic and the tuberculous types, in whom the blood pressure is low, show a diminution of the basophil cells in a majority of cases. . . .

"As to the relationship between the basophil cells of the pituitary and the suprarenals, 72.7 per cent. of the persons with suprarenals rich in lipoid (12 Gm. or more) show a marked increase of the basophil cells.

"Our studies on these two hundred and thirty-two pituitaries taken from normal and sick persons permit the conclusion that there is a definite relationship between the bosophil cells of the pituitary and the constitutional type of the patient; likewise between these and the blood pressure as well as the state of the suprarenals. In other words, there is a definite delationship between the basophil cells, the constitutional make-up of the patient, the blood pressure and the size of the suprarenals.

"There is, apparently, also a relationship between the basophil cells of the pituitary, the cholesterol content of the blood and the suprarenals. This is important because of the relationship between the cholesterol content of the blood and vascular disturbances."

According to Anselmino[17] and his associates, clinical as well as anatomopathologic observations indicate a relation between the anterior hypophysis and the pancreas. In acromegaly there is frequently a considerable increase in the pancreas, and in young diabetic patients a shrinkage of the eosinophil cells of the anterior hypophysis.

Chiari[18] reports a man who went into coma with a blood sugar of 642 mg. and died. At postmortem the pancreas was found to be essentially normal. The suprarenals were twice as large as normal. The hypophysis was of normal size. There was considerable hyperemia of the anterior lobe. "In the neurohypophysis, particularly in the basal areas attaching to the anterior lobe there were abundant basophilous cells."

In one series of 65 cases of diabetes, 18 showed adrenals weighing 18 grams or more.

Joslin[19] says: "The pituitary gland, according to Kraus, presents certain changes in its anterior lobe in diabetes which are nearly constant in young people. He noted a decrease in the number and size of the eosinophilic cells as well as their degeneration, and the weight of the gland was below normal."

Polycythemia or a polycythemic tendency is common in diabetes.[1h] Basophilism has a tendency toward polycythemia. Moehlig and Bates[16] showed that the pituitary is sometimes responsible for polycythemia.

Experimental Evidence. Drs. Houssay, Biasotti and Rietti[19] showed that large doses of anterior pituitary extract intraperitoneally injected

in normal dogs on a raw meat diet caused hyperglycemia, glycosuria and an increase in total content of ketone bodies in the urine.

Injections of anterior pituitary preparations cause diabetes in the pancreatectomized dog. They cause thirst and increased fluid intake in other experimental animals.

The anterior lobe is now credited with elaborating a diabetogenic, an adrenotropic and a thyrotropic hormone.[20] Dogs without pituitaries suffer degeneration of the adrenals, the gonads, the thyroid and the parathyroids. Dogs receiving pituitary preparations by injections or implantation show an increase in the activity of the adrenals, the gonads and the thyroid.

Raab[21] noted that the administration of pituitrin led to a decrease in the amount of fat in the liver. A fat metabolism hormone is claimed to have been isolated from the anterior lobe which increases the acetone bodies in the blood.[22]

Houssay[23] and his co-workers showed that removal of the pituitary, followed by removal of the pancreas, leaves the animal free from diabetes or having it in very mild form, but extremely sensitive to insulin. Implantation of the anterior lobe causes diabetes in these animals. Barnes[24] and Regan in Carlson's laboratory confirmed these results. Their dog could handle normal amounts of glucose without glycosuria.

Barnes, Regan and Nelson[25] showed that the administration of amniotin to female dogs prior to removal caused pancreatectomy to be followed by only mild glycosuria. When the injections of amniotin were stopped, the animals became severely diabetic. Resumption of the injections of amniotin caused a reduction in the glycosuria.

Summary. The pancreatic deficiency theory is inadequate. Evidence of pancreatic damage is usually lacking. Hyperinsulinism too usually fails to present evident changes in the pancreas. On the other hand, the association of diabetes with gigantism, acromegaly and tumors of the adrenals and various other clinical, experimental and pathological data very strongly suggest, if they do not actually prove, that insulin and the pancreas are but one side of a balance and that the usual cause of diabetes is an over-abundance of the pituitary and adrenal secretions.

Hypertension

At the present time as regards essential hypertension we are without hypotheses as to cause or hope as to cure of this condition. It is my belief that the evidence in the literature is practically conclusive as to the part played by the pituitary and adrenals in this condition. At the same time, evidence in the clinical literature justifies the hope that we have in radiotherapy an agent which will control and possibly cure many cases. The following pages are devoted to assembling some of the most important bits of evidence, particularly as to the cause of this condition.

Many years ago it was suggested that the excessive use of table salt had something to do with causing or maintaining hypertension. Salt restriction is still an orthodox measure in the treatment of hypertension. It has been shown that the adrenals are very influential in, if they do not actually control, sodium metabolism.[26] Harrop et al[27] have shown that cases of Addison's disease do very much better on a heavy intake of salt. Many patients with hypertension are heavy salt eaters.

Adrenal tumors are frequently associated with hypertension. French workers[28-29] are reported to have x-rayed the adrenals successfully for the relief of hypertension.

W. Landon Brown[30] states that in the earlier stages of acromegaly in association with hemianopia, lowered sugar tolerance and osseous hypertrophy, the blood pressure is high whereas in the latter stages with a high sugar tolerance, blood pressure is low.

Simmonds disease (destructive lesion of the anterior lobe) is associated with low blood pressure.

Toxic adenoma of the thyroid is frequently associated with hypertension. A thyrotropic hormone has been isolated from the anterior lobe of the pituitary.

Arn[31] has denervated the adrenals for the relief of hypertension.

Victims of hypertension are afflicted with headaches, the cause of which has never been explained. Measures which presumably influence pituitary function relieve the headaches.

Cushing's[25] cases of basophilism exhibited hypertension. In one case this was relieved by x-raying the pituitary.

The adrenals are dependent upon pituitary function as evidenced by:

The adrenotropic hormone isolated from the anterior lobe.

Degeneration of the adrenals after hypophysectomy.

Repair of this degeneration by pituitary transplants.

Herrick[32] describes the victims of hypertension in the same terms that an endocrinologist would use in describing an individual who had an abundance of pituitary and adrenal hormones.

Alvarez[33] says that the influence of sex is hardly recognizable in childhood; that it becomes exaggerated about the age of puberty and again in women about the menopause. "It is suggestive also that in young women there seems to be a high degree of correlation between the incidence of hypertension and the presence of the various symptoms and signs of ovarian hypofunction. It would seem that the internal secretion of the ovary is able in some way to suppress the tendency to hypertension which many of the girls presumably inherit equally with the men." Other writers[34] have expressed similar opinions. The sex hormone tends to suppress pituitary functions.

"Riesman[35] * * * emphasizes the importance of recognizing a variety of non-nephritic hypertension occurring often in women past middle life who are obese, undersized, ruddy complexioned and possessed of considerable mental and physical energy, without demonstrable arterial or renal abnormalities. The men of this group are apt to be deep chested, robust and great expenders of energy."

Goldzieher[36a] says: "The relation between the climacterium in women and hypertension is also striking and many theories have been built up on the conception that functional disturbances of the ovary and hypertension are closely connected. The frequency of hypertension in artifically sterilized women is particularly noteworthy and so is the coincidence of myoma and hypertension."

Fluhmann's[37] findings of an excess of the anterior pituitary sex hormone in women past the menopause helps to explain Goldzieher's observation.

Ten years ago Marañon[38] opined that the adrenals took on added activity at the menopause and that the body became more sensitive to adrenalin.

Some cases of hypertension are relieved by injections of corpus luteum. It is admitted by the experimentalists that corpus luteum depresses pituitary function.

Gutman[39] says: "During the past few years I have been able to conclude from the observations made in the study of women of the essential hypertension type that they were all in reality pituitropic individuals. * * * In others who suffered cardiorenal complications, the adrenal markings appeared to be predominating."

Goldzieher[36b] says:

"1. Phaeochrome tumors are apt to cause hypertension, vascular crisis, heart hypertrophy and vascular changes.

"2. In hypertension, particularly if arterio—or nephro-sclerosis has developed, usually large adrenals are present, which most frequently show characteristic gross and microscopic changes.

"3. The adrenalin content of the adrenals in hypertension is increased even if manifest enlargement should be absent.

"4. Essential hypertension is characterized in the beginning by its lability, based on a varying adrenalin output from as yet but functionally altered adrenals. If the hypertension becomes permanent, organic changes of the adrenals have been established already which make the hyperfunction of the adrenals a constant one.

"5. Other symptoms of increased sympathetic tonus, such as hyperglycemia, decreased glucose tolerance and glycosuria, are often prevalent in hypertension. The complication with diabetes is common. All this is well in keeping with the supposed adrenal hyperactivity.

"6. Cholesterolemia is another feature of hypertension. Adrenal function is instrumental in the regulation of cholesterol. Hypertensive arterial disease is characterized by cholesterol deposits, and on the other hand experimental cholesterol over-feeding will yield severe arteriosclerosis which is comparable to that in humans.

"7. All the factors which are known to be responsible for the etiology of genuine hypertension are also known as stimuli of adrenal activity.

"8. Adrenalin is the physiological stimulus of the sympathetic vasoconstrictor system and its cortical antagonist, cholin, is that of the depressor vagus system.

"9. Injection of adrenalin in sufficiently large and repeated doses is toxic and produces severe arterial changes. * * * Proper dosage will yield changes comparable to those in human arteriosclerosis.

"10. Retention of N waste products in the renal form of hypertension stimulates the adrenals, the function of which, as shown by N retention after adrenalectomy, is that of enhancing kidney excretion. This is achieved by the specific effect of adrenalin upon the efferent glomerular vessels. Hence the hypertension in renal insufficiency is of a compensatory nature and is attained through the mediation of the adrenals.

"11. Experiments have proved that resorption of kidney parenchyma, such as is supposed to occur in the so-called contracted kidneys, elicits adrenal hyperactivity and yields adrenal hyperplasia. Hence primary renal hypertension might change into or combine with essential hypertension."

Moehlig and Osius[40] fed their animals a high fat diet and injected pituitrin daily. Pronounced arteriosclerosis and marked hyperplasia of the adrenal cortex occurred in 100 days. The most frequent complication in diabetes is arteriosclerosis. Diabetics get a high fat diet. If they are receiving an excess of the pituitary hormone, their arteriosclerosis could be explained on the basis of this experimental evidence.

Cushing[2c] expressed the opinion that all known primary pituitary disorders cause marked secondary changes in the adrenal cortex.

Laroche[41] concludes that the cortical substance of the adrenal glands produces cholesterol and regulates its content in the blood.

Goldzieher[36c] says: "Autopsy findings in human hypertension on the other hand often reveal an increase in size of the cortex due to an excess of its lipoidal content."

"The intimate correlation between lipoid metabolism, adrenal cortex, and atherosclerosis is striking, although its mechanism is far from being clear. The cholesterol disturbance in atherosclerosis and its relationship to the changes of the adrenals has closed the ring of evidence which seems to prove that adrenal hyperfunction is among the most important factors in atherosclerosis.[36a] * * *

Chabanier and Truchot[42] report improvement in hypertension with functional and anatomical symptoms of dilatation of the left heart after treatment with a pancreas extract. This was an insulin-free extract which was isolated independently by the French and Germans. The mechanism for its action might easily be a suppression of pituitary and adrenal function.

Eisenberg and Wallerstein[43] say that hypertension is probably "the most important condition coexisting with pheochromocytoma. Of 53 cases 25 (47%) showed the association with hypertension. * * * While in some cases of tumor of the suprarenal cortex hypertension was continuous, in a large majority of the cases of tumors of the suprarenal medulla in which it was present, it was a paroxysmal type. * * *

"Peyron thought that the reason why hypertension is paroxysmal in the case of pheochromocytoma is that the intense proliferation and death of chromaffin cells interfere with the regular and complete secretion of epinephrine and that as a result of this, many pro-epinephrine and para-epinephrine particles get into the circulation and thus for the time being there is no hypertension.

"We are therefore confronted with the fact that in some cases of tumor of the suprarenal cortex and in most cases of those of tumor of the medulla * * * there is either paroxysmal-intermittent or continuous hypertension, and that very frequently removal of the tumor is followed by restoration of normal blood pressure."

Evelyn Rogers[44] cites a number of cases characterized by attacks of malaise, pallor, coldness and vasoconstriction of the extremities, sensation of epigastric constriction, with nausea and vomiting. During these attacks the blood pressure doubled. One man died with a clinical diagnosis of Graves' disease, chronic nephritis and hypertension. Autopsy showed normal thyroid and kidneys, generalized arteriosclerosis and a pheochromocytoma of the right suprarenal medulla. Another case with paroxysms of hypertension was relieved by radiotherapy to the lumbar region. He finally died in a condition resembling uremic coma. Autopsy showed a paraganglioma of the right suprarenal. The kidneys were normal.

In 1919 Neubauer[45] noted the association of hypertension and hyperglycemia and offered the theory that excessive activity of the suprarenals was the underlying factor. O'Hare[45] in this country noted that in one-fourth of the cases of hypertension without apparent kidney lesion there was definite hyperglycemia. He also noted that hyperglycemia was rare in cases of hypertension associated with a definite kidney lesion.

Kerppola[46] examined 31 cases of essential hypertension and found the sugar tolerance lowered in 80 per cent. and alimentary glycosuria present in 25 per cent.

Goldzieher[36e] says: "In brief the writer claims that Volhard's arterial spasm or Munk's undefined 'hematogenous' hypertension is nothing else but a symptom of overactivity of pressor endocrines and particularly of the paheochrome system. I wish to state emphatically that I do not identify hypertension with mere hyperadrenalinemia although adrenalin is the most powerful of the physiological pressor substances. There are other pressor hormones to be reckoned with, such as those of the pituitary and thyroid."

Cecil,[47] discussing hypertension, obesity, virilism and pseudo-hermaphroditism, says: "Of particular interest is hypertension. * * * It is not usually of the paroxysmal type but the blood pressure is constantly high. * * * Cortical tumors have been reported, particularly in men, in which the only symptom was hypertension."

Langeron and Loheac[48] conclude that primary tumors of the suprarenal gland may explain the pathogenesis of apparently primary or essential hypertension. One type consists of paroxysmal hypertension and is associated with paragangliomas. A second type of continuous hyperten-

sion may be caused by epitheliomas of the suprarenal cortex. Other writers[49-50-51] have commented on the relationship between adrenal tumors or hyperplasia and hypertension.

Treatment. It is believed that radiotherapy can restore to more nearly normal levels the function of the pituitary and adrenals. We have consequently used this agent in the treatment of a series of cases of diabetes mellitus and essential hypertension. Regard for the patient's welfare has caused us to proceed more cautiously than will be necessary when the various factors of dosage and frequency of repetition are fully worked out. In spite of this extreme caution our results have been not only interesting but hopeful. All but one case of hypertension have shown a considerable reduction in blood pressure with a coincident improvement in the patient's sense of well being. The diabetics have reported improvement in their sense of well being and the insulin requirement of some has been materially reduced.

Summary. At present we have no adequate hypothesis as to the cause of essential hypertension. The changes found in the pituitary in cases of hypertension, chronic nephritis and secondary contracted kidneys mentioned by Kraus and Traube, the occurrence of adrenal pathology in cases of hypertension and arteriosclerosis, the occurrence of identical sugar curves in diabetes and essential hypertension, the relief of hypertension by x-raying the adrenals, and other data point to hyperfunction of the pituitary and adrenals as the most likely cause of this condition and suggest a method of treatment.

The writer is conscious of the fact that this announcement may be regarded as premature. His defense against this charge is that if the theory and treatment are wrong, the more men who work on it, the sooner will the error be discovered and the entire matter be discarded. If it be correct, the more minds that are brought to bear on it, the sooner will the complete details be worked out and the treatment be made available to the general profession.

The writer is indebted to Drs. G. G. Dowall, Leroy Sloan, W. T. Harsha, E. C. Olson and R. H. Musick of the Illinois Central Service for their cooperation in this work.

BIBLIOGRAPHY

1. Joslin, Elliott P.; Treatment of Diabetes Mellitus, Lea & Febiger, Philadelphia 1928 4th Edition (a) p. 877; (b) p. 408; (c) p. 389; (d) p. 391; (e) p. 392; (f) p. 873; (g) p. 398; (h) p. 763.

2. Cushing, Harvey: Papers Relating to the Pituitary Body, Hypothalamus and Parasympathetic Nervous System, Charles C. Thomas, Springfield, 1932 (a) pp. 145-152; (b) pp. 168-172; (c) p. 158.

3. Anderson, K. W.: Diabetes Mellitus and Arteriosclerosis, Minnesota Med. 12: 484, Aug. 1929. Abst. JAMA 93: 875-876, Sept. 14, 1929.

4. Epstein, Albert A.: The Causal Mechanism of Diabetes Mellitus, JAMA 85: 29-31, July 4, 1925.

5. Boyd, William: Pathology of Internal Diseases, Lea & Febiger, Philadelphia, 1931, pp. 383-384.

6. Wüllenweber, G.: Diabetic Symptoms in Disorders of Thyroid and in Tumors of Suprarenals, Munchen Med. Wchnschr. 77: 144 Jan. 24, 1930. Abst. JAMA 94: 1186-1187, April 12, 1930.

7. Harris, Seale: Hyperinsulinism and Dysinsulinism, JAMA 83: 729, 1924.

8. Winans, H. M.: Hypoglycemic Convulsions with Hypoplasia of the Pancreas, Am. J. M. Sc. 185: 500-505, April, 1933.

9. Harris, Seale: Hyperinsulinism and Dysinsulinism (Insulogenic Hypoglycemia) Endocrinology 16: 29-42 Jan.-Feb., 1932.

10. Cushing, Harvey: The Pituitary Body and Its Disorders, J. B. Lippincott, Philadelphia, 1912.

11. Rowntree, L. G.: Diabetes and Dyspituitarism, Proc. Staff Meet. Mayo Clin. 2: 216-218 Sept. 14, 1927.

12. DeTakats, Geza and Fenn, G. K.: Bilateral Splanchnic Nerve Section in a Juvenile Diabetic, Ann. Int. Med. 7: 422-430, Oct., 1933.

13. Martin, Eric: Diabetes and Acromegaly, Rev. méd. de la Suisse Rom. 49: 693, 1929. Abst. J. Organotherapy 14: 296-297 Sept.-Oct., 1930.

14. White, Priscilla: The Potential Diabetic Child, JAMA 88: 170-171, Jan. 15, 1927.

15. Moehlig, R. C. and Ainslee, H. B.: Pituitary Gland and Cholesterol Metabolism, Ann. Clin. Med. 5: 772, 1927.

16. Moehlig, R. C. and Bates, G. S.: Influence of the Pituitary Gland on Erythrocyte Formation, Arch. Int. Med. 51: 207-235, Feb., 1933.

17. Anselmino, K. J., Herold, L. and Hoffman, F.: Pancreatropic Action of Extracts from Anterior Lobe of Hypophysis, Klin. Wchnschr. 12: 1245 Aug. 12, 1933. Abst. JAMA 101: 1277, Oct. 14, 1933.

18. Chiari, Hermann: A Case of Diabetes with Hypertrophy of the Suprarenal Capsule, Wien. med. Wchnschr. 42: 1318-1322, Oct. 10, 1929.

19. Houssay, B. A., Biasotti, A., and Rietti, C. T.: Correspondence dated March 1, 1933, from Buenos Aires, JAMA 100: 1444-1445, May 6, 1933.

20. Evans, Herbert M.: Present Position of Our Knowledge of Anterior Pituitary Function, JAMA 101: 425-432, Aug. 5, 1933.

21. Raab, W.: The Role of the Pituitary Posterior Hormone in Fat Metabolism, Endocrinology 14: 385-388, Nov.-Dec., 1930.

22. Anselmino, K. J. and Hoffmann, F.: Fat Metabolism Hormone of the Anterior Lobe., Klin. Wchnschr. 10: 2380, also 2383, Dec. 26, 1931. Abst. JAMA 98: 776 Feb. 27, 1932.

23. Houssay, B. A. and Biasotti, A.: Hypophysis, Carbohydrate Metabolism and Diabetes, Endocrinology 15: 511-523, Nov.-Dec., 1931.

24. Barnes, B. O. and Regan, J. F.: Relation of Hypophysis to Experimental Diabetes. Science 77: 214, Feb. 24, 1933.

25. Barnes, B. O., Regan J. F., and Nelson, W. O.: Improvement in Experimental Diabetes Following the Administration of Amniotin. JAMA 101: 926-927, Sept. 16, 1933.

26. Loeb, Robert F.: Mechanism of Decrease of Blood Sodium Chloride in Addison's Disease, Proc. Soc. Exper. Biol. & Med. Vol. 31, No. 1, 1933.

27. Harrop, G. A., Weinstein, A., Soffer, L. J. and Trescher, J. H.: The Diagnosis and Treatment of Addison's Disease, JAMA 100: 1850-1855, June 10, 1933.

28. Foreign Letters, JAMA 89: 705, Aug. 27, 1927.

29. Vaquez, Henri and Laidlaw, G.: Diseases of the Heart, W. B. Saunders, Philadelphia, 1924, pp. 461-462.

30. Brown, W. Landon, mentioned by Norris, Geo. Wm.:

Blood Pressure, Its Clinical Applications, Lea & Febiger, Philadelphia, 1917, 3d ed. p. 367.

31. Arn, Elmer R.: Personal communication.

32. Herrick, W. W.: Factors in the Prognosis of High Blood Pressure, Ann. Int. Med. 3: 467-476, Nov., 1929.

33. Alvarez, W. C.: Blood Pressure in 15000 University Freshmen, Arch. Int. Med. 32: 17-30, July, 1923.

34. Hoskins: Endocrinology and Metabolism, Edited by Lewellys F. Barker, D. Appleton & Co. New York, 1922, Vol. 1, p. 158.

35. Riesman, mentioned by Norris, Geo. Wm.: Blood Pressure, Its Clinical Applications, Lea & Febiger, Philadelphia, 1917, 3d ed. p. 278.

36. Goldzieher, Max A.: The Adrenals, The Macmillan Co., New York, 1929. (a) p. 240; (b) pp. 253-254; (c) 239; (d) pp. 250-251; (e) pp. 233-234.

37. Fluhmann, C. F.: Anterior Pituitary Hormone in the Blood of Women, Endocrinology 15: 177-183, May-June, 1931.

38. Marañon, G.: La Edad Critica, Soc. Espan. de Pub. Med. Madrid, 1919.

39. Gutman, Jacob: A Study of High Blood Pressure in Women from an Endocrine Standpoint, New York M. J. 114: 31-35, July 6, 1921.

40. Moehlig, R. C. and Osius, E. A.: The Pituitary Factor in Arteriosclerosis; Its Experimental Production in Rabbits, Ann. Int. Med. 4: 578-591, Dec., 1930.

41. Laroche, G.: Suprarenals and Cholesterol, Rev. franc. d'endocrinol. I: 185, July, 1923. Abst. JAMA 81: 1728, Nov. 17, 1923.

42. Chabanier, H. and Truchot, P.: Arterial Hypertension with the Symptom Complex of the Left Heart. Treatment by Means of a Pancreas Extract. Improvement. J.d'urol. 28: 405, 1929, Abst. J. Organotherapy 14: 304, Sept.-Oct., 1930.

43. Eisenberg, A. A. and Wallerstein, Harry: Pheochromocytoma of the Suprarenal Medulla (Paraganglioma) Arch. Path. 14: 818-835, Dec., 1932.

44. Rogers, Evelyn: Paroxysmal Hypertension Associated with a Ganglioneuroma of the Suprarenal Medulla. Am. Heart J. 8: 269-274, Dec., 1932.

45. Herrick, W. W.: Hypertension and Hyperglycemia, JAMA 81: 1942-1944, Dec. 8, 1923.

46. Kerppola, W.: Essential Hypertension, Acta med. Scandinav. 57: 515, Feb. 22, 1923. Abst. JAMA 80: 1888, June 23, 1923.

47. Cecil, Howard L.: Hypertension, Obesity, Virilism and Pseudohermaphroditism, as Caused by Suprarenal Tumors, JAMA 100: 463-466, Feb. 18, 1933.

49. Pincoffs, M. C.: Paroxysmal Hypertension Associated with Suprarenal Tumor, JAMA 93: 63, July 6, 1929.

50. Galata, G.: Grave Climacteric Hypertension Treated with Unilateral Suprarenalectomy, Riforma med. 45: 1449, Oct. 26, 1929. Abst. JAMA 94: 225, Jan. 18, 1930.

51. Meyer, Jacob and Frumess, Gerald: Tumors of Suprarenal Gland with Special Reference to Carcinoma of Cortex; Report of Case, Arch. Int. Med. 48: 611-626, Oct., 1931.

THE TREATMENT FOR GENERAL PARESIS BY MEANS OF THE ELECTRIC CABINET, ARSENICALS AND TYPHOID VACCINE*

EMIL T. HOVERSON, M. A., M. D.

AND

GEORGE W. MORROW, M. D.

KANKAKEE, ILL.

In a preliminary report[1] the use of an electric cabinet in the treatment for general paresis was described. To date over five hundred treatments by the use of the cabinet have been given with results which encourage us in the belief that this form of treatment is a valuable and efficient method of treating general paresis, and the allied conditions, such as central nervous system syphilis.

A description of the cabinet according to the original report follows: "The cabinet is constructed of 24 gauge galvanized iron. The outside dimensions of the cabinet are: length 60 inches, width 28 inches, and the center height is 26 inches. The center inside height is 24 inches, and the sides are vertical for a height of 14 inches, and then curved gradually to the center. One end of the cabinet is closed by a galvanized iron end section "double-sealed" to the main body of the cabinet. This provides additional construction strength and also aids in conserving the heat generated. The other end of the cabinet is open, and has a ¼ inch rod rolled into the outside of the cabinet. This end is so arranged that the blanket draped about the shoulders and neck of the patient may be fastened by means of clamps attached to the curved rod.

"For the sake of smoothness and strength of construction the bottoms of the sides and the end are rolled inwards in a one inch roll. Inside of the frame are three grooved, wooden ribs which follow the curve of the cabinet. The end ribs are placed 10.5 inches from the ends and the center rib 19.5 inches from each end rib. Wires insulated with asbestos are placed in the grooved ribs and lead to the electric sockets which are located on the sides of each rib. Four such sockets occur on each side of the ribs, giving a total of eight lights per rib, and a total of twenty-four lights. The lights are placed in a series of twelve each, which permits the use of a greater amount of heat in producing the desired temperature and of a lesser amount of heat to maintain a given temperature with comfort to the patient. The lights are controlled by two switches on the outside of the cabinet, one for each series of lights."

The only alteration which has been made to the original cabinet consisted in moving the switches controlling the electric lights from the top to the side of the cabinet. This change facilitated the manipulation of the control switches.

Although no exact routine for the treatment of every patient can be formulated, the following

*From the Kankakee State Hospital.

procedure is the one generally used in starting treatment on a new patient, whose reaction to external heat is not known. The cabinet is so constructed that it rests on the hospital bed, and can be lifted on or off the bed at will. Prior to the placing of the cabinet on the bed, the bed is prepared by placing a rubber sheet over the mattress, and placing cotton sheets over this.

The patient is placed on the bed, completely undressed, and covered only with a cotton sheet, and then the cabinet is placed over the patient, and it rests on the bed. Five or six woolen blankets are laid over the cabinet and another blanket is draped snugly over the open end of the cabinet, and about the shoulders and neck of the patient.

All of the lights in the cabinet are turned on and kept on until the patient's axillary temperature reaches 101 F. degrees. Then one-half of the lights are turned off. When the temperature reaches 102 degrees, the remaining twelve lights are turned off. The patient remains in the cabinet just as if the lights were on, and in practically all cases the axillary temperature rises to 103-104 degrees, and stays at this level for several hours. When the temperature recedes to 101 degrees, the cabinet is removed and the patient is placed in a dry bed. All temperatures are axillary and are taken every fifteen minutes during the treatment and until the patient's temperature has dropped to 101 degrees. Thereafter temperature readings are taken every half hour until a normal reading is reached.

In practically all cases a temperature of 102 degrees is reached in one and one-half hours, but a variation of a half hour either way is observed in some cases.

The first treatment is started at eight o'clock in the morning, and the temperature is 102-103 degrees, and the lights turned off at about ten o'clock A. M. Usually the height of the patient's temperature is reached at noon, and following this there is a gradual decline and at about two o'clock in the afternoon the patient is placed in a dry bed, and by five the temperature is back to normal.

During the first treatment, as well as in subsequent ones, the patient's reactions are closely watched and the procedure for other treatments is planned to suit individual responses.

The second treatment is carried out in a similar manner to the first, except that one-half of the lights are turned off at 102 degrees, and all when the temperature of the patient reaches 103 degrees.

The patients who responded satisfactorily during the first and second treatments, i. e., were quiet and cooperative, had a temperature reading of 103-104 degrees in two to three hours, and whose temperature did not rise over 105 degrees, are given all subsequent treatments according to the following procedure: All of the lights are kept on until the patient's temperature reaches 104 degrees and then half of the lights are kept on until the temperature is 105 degrees. The temperature of the patient continues to rise to 106-106.4 degrees and remains at this level for one to two hours. Then the temperature is allowed to fall gradually and usually within three hours after it has receded below 106 degrees it is 103 degrees. The patient is placed in a dry bed, as originally described.

If it is desired to maintain the temperature of 106 degrees for a longer time, twelve of the lights are turned on when there occurs a drop of .2=.5 degrees and they are kept on until the temperature again rises a fraction of a degree. This procedure may be repeated as often as is compatible with the patient's condition.

All temperature readings are axillary. If any reading seems questionable it is at once checked by a rectal temperature reading. In all cases the axillary temperature is checked by a rectal reading when the temperature is 106 degrees.

A light breakfast is served in the morning. During the treatment water is given freely, and often as high as four liters are consumed. Perspiration is always profuse. The cessation of perspiration is a warning sign of heat prostation, but as yet this has not occurred in the treatments. Should this occur, the patient should at once be placed in a dry bed and treated as the occasion demands.

Some patients become restless and turn from side to side when their temperature readings are at the highest point. Since no harm can result from this, the only precaution necessary is to see that the blankets remain draped about the shoulders. In our experience practically all of the patients cooperated willingly after the fifth treatment. It is not at all unusual to have patients ask for a treatment.

The foregoing procedure has been followed in practically all of the cases treated but it was necessary to modify it for uncooperative, restless patients. In these cases a nurse or attendant was in constant watch and when necessary, held the patient's shoulders while he was in the cabinet. When a patient is restless his temperature usually rises more rapidly than when he is quiet. Hence, there is no need to say that he must be carefully watched. In a few instances, where the patient was exceptionally excited, restless or confused, it was deemed advisable to administer from one to six injections of typhoid vaccine before starting the regular cabinet treatments.

From our experience[1] with typhoid vaccine, we can definitely state that very little lasting improvement can be expected from less than twenty injections of typhoid vaccine. Therefore, any improvement in the patient's condition that was noted after a course of cabinet treatments cannot be ascribed to the results following the initial typhoid vaccine injections.

Also in certain cases, patients were in a poor physical condition on admission. We have pointed out[2] that the physical health improves following typhoid vaccine injections. Such patients were also given from two to ten typhoid vaccine injections, and following this they were put on regular cabinet treatments.

In no case has it been necessary to administer a sedative drug.

The usual course of cabinet treatments consists of from twelve to twenty treatments. These are given twice a week for the required number of times. Following this fever therapy, arsenicals or mercury are given weekly for ten weeks. Since January, 1933, the usual arsenical has been in the form of tryparsamide and this is given wherever possible.

If the desired improvement has not been reached following the arsenical or mercury injections, a second course of fever therapy is started.

The patients at the Kankakee State Hospital are classified according to the Statistical Handbook, published by the Department of Public Welfare, State of Illinois, Second Edition.

The following table shows the number and kind of treatments with the classification of the patient and the results of the treatment:

Case Number	No. of Typhoid Injections	Neosalvarsan .9 Grams Dose	Murcury .2 Grain Dose	Tryparsamide	Electric Cab. Treatments	Hours Over 105 Degrees	Classification	Results
1.	10	6	0	10	26	40.5	Gen. Paralysis, Cerebral	Paroled
2.	8	16	16	1	14	15	Psy. Cerebral Syphilis	Paroled
3.	10	6	0	10	19	16	Gen. Paralysis, Cerebral	Paroled
4.	10	4	0	0	16	30.2	Gen. Paralysis, Tabetic	Marked Improvement
5.	14	8	0	10	18	32.2	Gen. Paralysis, Cerebral	Improved
6.	10	8	0	0	12	20	Gen. Paralysis, Tabetic	Paroled
7.	10	1	0	10	10	29.5	Gen. Paralysis, Cerebral	Paroled
8.	2	0	0	10	10	18.2	Psy. Cerebral Syphilis	Paroled
9.	22	16	11	10	14	20	Gen. Paralysis, Cerebral	No Improvement
10.	1	0	0	0	10	18	Gen. Paralysis, Cerebral	Paroled
11.	2	0	0	10	15	27	Gen. Paralysis, Cerebral	Improved
12.	0	0	0	10	15	27	Gen. Paralysis, Cerebral	Paroled
13.	0	0	0	10	12	16.5	Gen. Paralysis, Cerebral	Paroled
14.	0	0	0	8	17	20	Gen. Paralysis, Cerebral	No Improvement
15.	0	0	0	10	13	24	Gen. Paralysis, Tabetic	Paroled
16.	16	8	8	0	10	17	Gen. Paralysis, Tabetic	No Improvement
17.	16	8	8	10	15	17.7	Gen. Paralysis, Cerebral	Worse
18.	0	0	0	10	16	22	Gen. Paralysis, Cerebral	Paroled
19.	6	0	0	3	15	26.5	Gen. Paralysis, Cerebral	Improved
20.	0	0	0	3	15	40.0	Gen. Paralysis, Cerebral	No Change
21.	0	0	0	10	15	32	Gen. Paralysis, Cerebral	Paroled
22.	0	0	0	3	13	27	Gen. Paralysis, Cerebral	Improved
23.	0	0	0	10	13	19.5	Gen. Paralysis, Cerebral	No Improvement
24.	24	8	8	0	10	19.5	Gen. Paralysis, Cerebral	No Improvement
25.	34	16	16	3	16	15	Gen. Paralysis, Cerebral	Paroled

Cases No. 16 and 17 were put on the cabinet treatments at the insistence of relatives. They had shown no improvement following a series of typhoid injections. In cases 24 and 25 treatment was given following the failure of typhoid vaccine to show any change after a stay here of one year. Case 25 immediately showed a marked improvement and this was continued up to the time he left this hospital.

Of the twenty-five patients treated by this method or combined method with typhoid vaccine and tryparsamide, thirteen or 52.0% were represented to the medical staff, and all were recommended for parole.

The remaining twelve patients in this series have not been presented before the medical staff because they have either not shown enough improvement to warrant their parole or because they have gradually become worse or have shown no essential change.

Of the thirteen patients recommended for parole by the medical staff, eight have left the institution and are making a good adjustment on the outside. The remaining five patients all have a ground parole here and are waiting for some friend or relative to whom they may be paroled.

Six patients showed no change or became worse and the remaining six have shown improvement, which, if it continues, may warrant their representation with view of their release in the near future.

The results obtained by this method of treating paresis compare favorably with the results using other forms of producing the hyperpyrexia. This method has the added advantage that the cost of the cabinet is small and they last for years (the cabinets have been in daily use eight months and are in good condition). Further, in this form of treatment, the patient is free to move about as he wishes, and is not burdened by countless covers.

CONCLUSIONS

1. Twenty-five male patients have been treated by means of the electric cabinet, arsenicals and typhoid vaccine.

2. Of the twenty-five patients so treated 13 have shown sufficient improvement to warrant their release. This expressed in percentage is 52.0% of the patients treated.

3. Apparently it is the hyperpyrexia, and not the way it is produced, that is the beneficial agent in paresis.

4. There are certain cases of paresis which show no improvement in spite of intensive antiparetic treatments.

REFERENCES

1. Hoverson, E. T., Morrow, G. W., and Hawthorne, R. O.: Treatment of General Paresis by Typhoid Vaccine and the Electric Cabinet. Ill. Med. Jour. Vol. LXIII, 252, March, 1933.

2. Hoverson, E. T., Morrow, G. W., and Hawthorne, R. O.: The effect of Typhoid Vaccine on the Erythrocyte count in Dementia Paralytica. Ill. Med. Jour. Vol. LXII, 465, November, 1932.

A DIATHERMY ELECTRODE BASED ON ENTIRELY NEW PRINCIPLES

H. E. KIMBLE, M. D.,
Clinical Assistant
H. J. HOLMQUEST, B. S. (M. E.)
Lecturer in Applied Physics, Department of Physical Therapy, Northwestern University Medical School
CHICAGO

High frequency electric currents used for medical diathermy are applied to tissues in which heat is to be generated by means of conductive plates called electrodes. Obviously the material of which these electrodes are made must have certain definite properties in order that the administration of medical diathermy be satisfactory. It is important that uniform electrical contact between the electrode and the skin of the patient be secured. To obtain readily such contact, the electrode must be highly flexible, especially when the part of the body to be treated has an irregular anatomical topography. Furthermore, this close apposition of electrode and skin must be secured without the application of excessive pressure. If an electrode does not make uniform contact, there will be areas of high concentration of current and resultant excessive superficial heating at these points. If uniform contact can be secured only by exerting a high pressure, the higher areas of tissue will obviously be subjected to greater pressure than will the lower areas. This non-uniform pressure exerted on the tissues will impair circulation at the points of excessive pressure and tend to prevent the desirable vasodilatation which is so marked an effect of medical diathermy. Furthermore, the exertion of non-uniform pressure on the tissue will modify the flow of current

and may result in excessive superficial heating at certain points.

Obviously, perfect physical contact between electrode and skin is not alone sufficient for the

FIG. 1

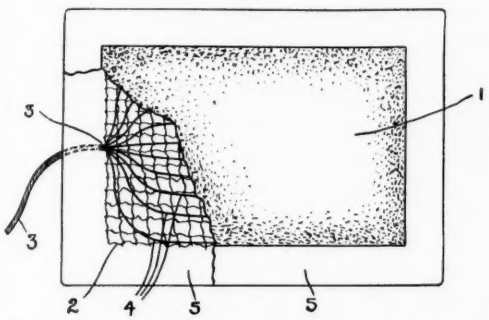


Fig. 1. Diagram showing construction of the new diathermy electrode. 1. Flexible, conducting electrode surface. 2. Specially woven metallic mesh. 3. Stranded conductor. 4. Strands of conductor making multiple contact with mesh. 5. Resilient backing of electrode.

best results. Perfect physical contact must of course be secured but it must be secured with uniform pressure on the tissues. It is also necessary that the conductivity of the electrode, which is electrically in series with the tissues of the patient, be greater than that of the skin and superficial tissues. Otherwise, the electrode would heat more rapidly than the superficial tissues on

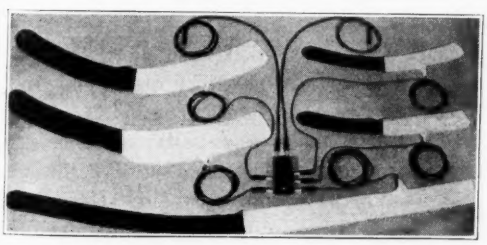


Fig. 2. Electrodes for the producton of electro-pyrexia by the belt and cuff method

the application of an electric current and before the tissues had received an adequate treatment would attain a temperature sufficiently high to burn the patient.

Another requirement of a satisfactory electrode is that no cracks develop in the electrode and that it have no sharp edges or projections. Such imperfections result in concentration of current and the excessive local heating ordinarily referred to as "hot spots." Together with the characteristics already discussed, the electrode must be durable and capable of extensive use.

Various electrodes have been designed and put on the market for diathermy application. There, however, has been no marked departure from the type used a quarter of a century ago. In general, the material of which diathermy electrodes have been constructed has been a more or less flexible metal foil. An attempt has been made to facilitate the moulding, when applied, of the foil electrode more closely to the surface of the body by perforating the foil and sinuating the periphery of the electrode. The procedure has been successful to a certain degree—in fact, this design has made available the most satisfactory foil electrode and the most satisfactory of all diathermy electrodes up to the present time.

The authors, early in their investigation of electrode design, realized that solid metallic electrodes could not be made more flexible than had

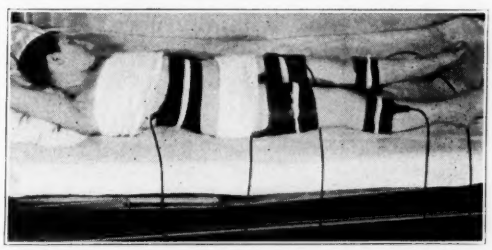


Fig. 3. Illustrating the application of electrodes for electro-pyrexia by the belt and cuff method, a method which has been successfully employed by Dr. Arthur C. Jones of Portland, Oregon

already been done by Neymann and Osborne in their design of electrodes for electro-pyrexia and still withstand hard usage. Therefore, attention was directed to research on a new type of conductive material which would have the desired flexibility. After extensive investigation and experimentation, a material possessing the desired physical and electrical properties was produced. This material consists essentially of a mixture of an adhesive substance and a highly conductive metallic powder in such proportions as will give maximum conductivity with maximum flexibility. While yet in the plastic state, this material is formed into electrode surfaces of the required size and shape. After these surfaces are processed, the material becomes highly conductive and flexible. During processing, the

electrode surfaces become securely and permanently fixed to a metallic mesh. This metallic mesh is specially woven so as not to impair the flexibility of the electrode surface. Multiple electric contact with the mesh is made by means of a "fanned out" stranded conductor. The entire electrode is backed with a highly resilient, flexible rubber. The electrode in its finished state consists of a lead-in conductor, rubber backing, current distributing mesh, and conducting surface, permanently assembled into a single compact unit.

The salient characteristics of this electrode are:

1. High Flexibility.
2. Requisite conductivity.
3. Durability.

Use of this electrode with an appropriate electrode compound should materially reduce the possibility of "hot spots" and diathermy burns. An electrode of such high flexibility is easy to apply, perfect apposition with skin being secured without the application of excessive pressure. It has no sharp metallic edges and projections to cause high concentration of current and resultant burns. If, after very long or careless use of the electrode, its surface should crack, no arc will be established between the separated portions of the surface because the underlying conducting mesh will keep the entire electrode surface and all parts of it at the same electric potential.

ROENTGEN VISUALIZATION OF THE BILIARY TREE FOLLOWING A BARIUM MEAL*

HARRY A. SINGER, M. D.
AND
DAVID H. WAGNER, M. D.
CHICAGO

Visualization of the intrahepatic bile passages is readily accomplished in the presence of an external biliary fistula by simple injection of a contrast medium. Bismuth paste as used by Tenney and Patterson or iodized oil which is generally preferred yields upon injection, striking roentgenograms of the biliary tree. In the absence of a biliary fistula the intrahepatic radicals are only occasionally visualized. The outlining of the bile ducts under this latter condition is the result of the retrograde passage of either air or barium or both due most frequently to the presence of an internal biliary fistula. The visualization with barium will be treated at some length later. The demonstration of the biliary tree with air is quite rare as only three examples could be found in medical publications. Brief mention of the individual cases might therefore prove of interest. Ohnell and Lindbloom in a routine cholecystographic examination observed a tree-like system of rarefactions within the liver shadow. This phenomenon was apparently due to a spontaneous cholecysto-duodenal fistula which permitted the entrance of air into the tributary channels of the hepatic ducts. A somewhat similar case of air in the bile passages demonstrated by x-ray was reported by Friederich in a patient who had an artificial choledocho-duodenostomy. Air injection of the bile passages was reported also by Graeberger. The unique observation of Biesi although not actually involving the intrahepatic divisions might be appended here. Biesi noted following a contrast meal administered to a patient who had suffered from cholelithiasis, air and barium in the gallbladder due to a spontaneous cholecysto-duodenostomy.

A survey of the literature yields reports of visualization of the biliary passages by means of barium taken by mouth in 24 cases. In 7 of the 24 instances an artificial communication had been previously established by operation. In 5 (Lenk, Swalm and Marges, Fetzer, Parade and Westphal) of the 7 cases with anastomoses a choledocho-duodenostomy had been performed. In the other two operative patients (Graeberger, cases 2 and 3) the duodenum had been united to the gallbladder. Of the remaining 17 instances of visualization of the biliary tract by barium 8 were due to spontaneous internal fistulae and 5 to an insufficient papilla (Beall and Jagoda, Jenkinson and Browse, case 2, Venable and Briggs, Stephenson and recently Rees). In four reports (Reiman, Davis, 2 cases, Fishbaugh) the explanation of the retrograde passage of barium into the bile channels was not apparent. Of the 8 spontaneous fistulae 4 were between the gallbladder and duodenum, 3 between the common duct and the duodenum and one between the gallbladder and the stomach (Dalsace). In

*From the University of Illinois College of Medicine, Department of Medicine and the Cook County Hospital.

the group of spontaneous cholecysto-duodenostomies one was occasioned by a ruptured ulcer (Harding). In the other three cases (Habbe and Smith, Carman and Miller, Jenkinson and Browse, case 1) the cause of the union between the two organs was not stated. In two of the three instances of anastomosis between the common bile ducts and duodenum the communication was due to perforation of a peptic ulcer (Kantor and Jaffin, case 2, Bignami and Agati). The cause of the fistula in the other case of choledocho-duodenostomy also reported by Kantor and Jaffin (case 1) apparently was not established. To the above series of 24 cases of visualization of the biliary tree by barium can be added one which we observed.

Case Report. M. A., a white male of 59, consulted us in May, 1929, because of upper abdominal pain. With the aid of the patient's son, a recent graduate in medicine, the following account of the previous history was obtained. Early in June 1927, the patient developed a silent, progressive jaundice, obstructive in type, associated with the presence of a distended gallbladder. At no time did the obstruction become complete as bile persisted in the stools. On July 6, 1927, operation was performed and disclosed a dilated common duct, a distended gallbladder, a normal pancreas and a few soft, pink, peripancreatic lymph glands. In probing the common duct an obstruction was encountered just proximal to the ampulla of Vater. The surgeon considered the lesion to be a benign stricture of the duct. A cholecystectomy and a choledocho-duodenostomy were performed. So far as could be ascertained the gallbladder was normal and for this reason the logic of its removal could not be understood by us. Following operation the jaundice rapidly disappeared and the patient made an uneventful recovery.

For approximately six months subsequent to the operation the patient felt well except for a sense of epigastric fullness following ingestion of a fair-sized meal. The discomfort would be relieved by belching but this was as a rule difficult to accomplish. After the first half-year the epigastric fullness became much more troublesome inasmuch as distress followed the intake of even small quantities of bland food. Approximately nine months following operation the patient began to experience in addition to the fullness mentioned above, severe pain localized to the right upper quadrant. The pain which was of a colicky nature would occur during or within a few minutes after meals and often was of such severity as to render the patient speechless. The patient would as a rule arise from his chair and walk about doubled over until the pain subsided. This usually required but a few minutes. Recently this sharp pain increased in intensity and frequency to such an extent that the patient was fearful of eating and as a result was unable to perform any physical work. The patient had consulted two surgeons and two internists prior to visiting us and had been fluoroscoped twice. No explanation of the symptoms was offered, although the patient's son was told there was some delay in the emptying of the stomach due to a low-grade duodenal obstruction.

In listening to the patient's complaints one could hardly avoid being reminded of the clinical manifestations of gallbladder disease. The fullness after meals, the sensation of a weight in the epigastrium and the desire to belch differed in no way from the dyspepsia seen in patients with cholecystitis or cholelithiasis. The sharp pain of short duration occurring in the upper right quadrant resembled abortive biliary colics. The idea that the basis of the symptoms was to be sought in the biliary tract immediately suggested itself. Knowing that there was some impediment to the escape of food through the duodenum it seemed reasonable to assume that the gastro-duodenal chyme might be forced by peristalsis into the anastomosed portion of the common duct. With this possibility in mind an x-ray examination with barium was recommended and carried out by Dr. C. H. Warfield.

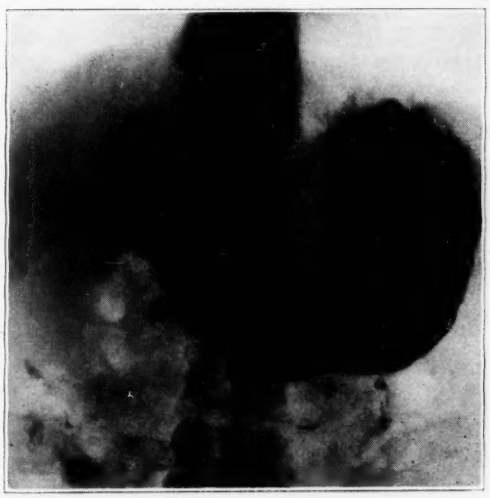


Fig. 1. Roentgen Visualization of the intrahepatic ramifications of the biliary system following administration of a barium meal. A choledocho-duodenostomy had been performed for an obstruction at the ampulla 22 months prior

It required only a moderate amount of compression of the stomach and distal duodenum to cause the contrast medium to enter the choledochus and ascend into the intrahepatic bile passages. The injection of the biliary tract with barium seen fluoroscopically is well demonstrated in the accompanying illustration (Fig. 1). A moderate stenosis of the descending portion of the duodenum was also established. A gastro-enterostomy and exclusion of the pylorus by ligature with kangaroo tendon was recommended. On August 13, 1929, operation was performed by Dr. Karl A. Meyer, who found

the duodenum, the common bile duct and the omentum bound together into an inextricable mass. It was deemed inadvisable to attempt to separate the individual structures by dissection. A posterior gastro-enterostomy was performed and the pylorus allowed to remain patent, as it also was covered by adhesions and was not readily accessible. No masses were palpable in the region of the head of the pancreas or the ampulla. The lymph glands were grossly free from malignant invasion. The recovery was an uneventful one. Within two weeks the patient left the hospital free of symptoms. He soon regained his weight and strength and returned to his former occupation. An x-ray examination performed six weeks after operation showed that practcally all the barium passed through the gastro-enterostomy stoma. None was seen to leave the duodenum. Barium could no longer be forced into the common duct. The patient remained in a state of comparatively good health until April, 1930, when he again developed abdominal pain. The pain became increasingly worse and on this account in May, 1931, exploration was undertaken elsewhere. A diffuse thickening with induration of the pancreas was found but no metastases. The patient died in September, 1931, with the clinical picture of an abdominal carcinoma.

Commentary. Since the time we observed the passage into the intra-hepatic ducts of barium given by mouth we have made a routine search for similar cases. Although our material has included several individuals previously subjected to choledocho-duodenostomy and similar operations no additional instance of biliary tree visualization presented itself to date. We have been led to conclude therefore, that the retrograde filling of the bile channels occurs only in the presence of certain exceptional conditions. What these conditions are cannot be definitely determined. In most of the reported cases of biliary tree visualization no information other than the presence of a fistula is offered to explain the regurgitation of barium into the liver. Since the retrograde passage of duodenal contents into the liver occurs only exceptionally even where an internal biliary fistula exists the mere presence of the preternatural communication does not of itself explain the altered physiology. An additional factor is suggested by the study of our case, viz., the interference with duodenal emptying. That the obstructive element was of significance is borne out by the fact that following gastro-enterostomy the barium failed to enter the liver. In those cases also in which an internal biliary fistula is absent and the retrograde passage is ascribed to an incompetent sphincter of Oddi, intraduodenal pressure occasioned by obstruction is apparently an important factor. In the case of Venable and Briggs a high intestinal obstruction producing an increase of pressure within the duodenum appeared to result in regurgitation into the common and hepatic ducts. In the case of Rees the explanation for the reflux was given as traction by a fistulous tract resulting in incompetence of the sphincter of Oddi. However, at operation the duodenum was found to be dilated and the tract was found to cross the pars horizontalis apparently producing a partial obstruction.

Visualization of the biliary ramifications following ingestion of a barium meal is generally an accidental observation. The contrast medium is administered as a rule for the purpose of disclosing an intrinsic gastric or duodenal lesion. The presence of an internal biliary fistula unless artificially produced is not suspected and the injection of the biliary system comes as a great surprise. The symptoms which occur in the presence of regurgitation into the biliary passages are not uniform. Most frequently they resemble the manifestations of gallbladder disease. At times as in the case of Beall and Jagoda a cholangitis develops indicated by the presence of chills and fever. Jaundice as one would anticipate from the knowledge of conditions present is exceptional. It was absent in all the barium cases and present in only one of the cases in which air was noted in the intrahepatic ducts (Ohnell and Lindbloom). Occasionally the symptoms are constitutional and do not suggest the location of the difficulty. For instance, in the case described by Dalsace of spontaneous cholecysto-gastrostomy, asthenia and emaciation were the presenting complaints. The occurrence two years prior, however, of abdominal crises and jaundice might have suggested a point of attack. As seen from the above description none of the symptoms associated with retrograde filling of the biliary tree are distinctive. The diagnosis can hardly be made in the absence of a history of an artificial or spontaneous internal fistula without the aid of the x-ray. With the knowledge of a previous anastomotic operation the presence of symptoms of the gallbladder type is highly suggestive. When as in our case it is known also that the gallbladder is absent the

occurrence of selective dyspepsia and upper abdominal colics permit a presumptive diagnosis.

SUMMARY

1. In the absence of an external biliary fistula occasional roentgen visualization of the intrahepatic biliary tree is encountered due to barium or air or both.

2. The retrograde passage of barium and air occurs in connection with an internal biliary fistula or an incompetent sphincter of Oddi.

3. An internal biliary fistula or incompetent sphincter does not of itself explain the regurgitation into the hepatic ramifications. Increased intraduodenal pressure due to obstruction is apparently an additional requisite factor.

4. The symptoms associated with retrograde filling of the biliary channels resemble those of gallbladder disease and its complications.

5. The diagnosis prior to x-ray examination can be suspected when an internal biliary fistula (usually operative) is known to exist.

1819 West Polk St.

BIBLIOGRAPHY

1. Beall, F. C., and Jagoda, S.: Injection of the Bile Ducts with Barium, J. A. M. A., 76:1483, 1921.

2. Biesi: Referred to by Graeberger (10).

3. Bignami, G., and Agati, D.: Barium Injection of Bile Ducts Through Choledocho-Duodenal Fistula During Diagnosis of Perforated Duodenal Ulcer, Case, Radiol. med., 18:545 565, 1931.

4. Carman, R. D., and Miller, A.: The Roentgen Diagnosis of Diseases of the Alimentary Canal, B. W. Saunders Co., Phil. P. 369, 1917.

5. Dalsace, J.: Observation de fistule cholecysto-duodenale, Arch. d. mal. de l'app. digestif., 20:194-199, 1930.

6. Davis, L.: Reflux of Duodenal Contents Through the Common Bile Duct, New England J. Med., 200:313, 1929.

7. Fetzer, H.: Kontrastbrei in den Gallenwegen als diagnostisches Merkmal, Röntgenpraxis, 2:308-312, 1930.

8. Fishbaugh, E. C.: Referred to by Kantor and Jaffin (14).

9. Friederich, L. V.: Luft in den Gallenwegen als diagnostisches Merkmal, Fortschr. a. d. Geb. d. Röntgenstrahlen, 39:616, 1929.

10. Graeberger, G.: Beitrag zur Röntgendiagnose innerer Gallenfisteln, Acta radiol., 12:164-174, 1931.

11. Habbe, E., and Smith, L. A.: Unusual Bile Duct Visualization by Roentgenogram of Barium Meal, J. A. M. A., 86:476-478, 1926.

12. Harding, D. B.: A Roentgen Study of the Lesions of the Stomach and Duodenum with an Analysis of the Errors in Diagnosis, Am. J. Roentgenol., 22:36-42, 1929.

13. Jenkinson, E. L., and Browse, I. E.: Visualization of the Bile Ducts Following the Administration of a Barium Meal, Am. J. Surg., 12 (N. S.): 499-501, 1931.

14. Kantor, J. L., and Jaffin, A. E.: Roentgen Visualization of the Bile Ducts with Special Reference to Internal Biliary Fistulae, Radiology 10:10-15, 1928.

15. Lenk, R.: Berichte aus den Wissenschaftlichen Vereinen. Röentgenologische Darstellung des Gallengangsystems, Wien. med. Wchnschr., 75:1594, 1925.

16. Ohnell, H., and Lindbloom, K.: Air Filled Bile Ducts in a Case of Fistula Between the Doudenum and Common Bile Duct, Acta radiol., 10: 121, 1929.

17. Parade, G. W.: Kontrasfüllung des Gallengangsystems gelegentlich einer Magen-Darmdurchleuchtung bei Anastomose zwischen Gallenblase und Zwölffingerdarm, Röntgenpraxis, 2:366-368, 1930.

18. Rees, C. E.: Duodenocolic Fistula with Incompetent Sphincter of Oddi. J. A. M. A., 100:496-497, 1933.

19. Reiman: Spontane Darstellung des Chloedochus, Fortschr. a. d. Geb. d. Röntgenstrahlen, 41:802. 1930.

20. Stephenson, F. B.: Opaque Meal in the Liver Ducts, J. Radiol., 2:35-40, 1921.

21. Swalm, W. A., and Marges, W. F.: Visualization of the Hepatic Ducts and Common Duct with Choledochus Stasis, Am. J. Surg., 7:521-525, 1929.

22. Tenney, C. F., and Patterson, S. H.: Injection of Bile Ducts with Bismuth Paste and Observations on the Flow of Bile, J. A. M. A., 78:171-173, 1922.

23. Venables, J. F., and Briggs, P. J.: Visualization of Bile Ducts After an Opaque Meal, Guy's Hosp. Rep., 79:123-126, 1929.

THE INNOCENT BYSTANDER*

J. P. SIMONDS, M. D.

CHICAGO

The newspapers record almost daily the fate of an innocent bystander. Without thought of danger and without warning of its presence, a man finds himself in the line of gunfire in a battle between police and gangsters or between two rival organizations of racketeers; or, while standing on the sidewalk or a safety island, he is suddenly run down by a careening automobile. His body may be mangled by an airplane which plunges down upon him out of the blue sky like Jove's thunderbolt; or his head may be crushed by brick or flower-pot falling from an upper story. Some fate brings the innocent bystander and the bullet, the automobile, the airplane or the flower-pot together just at the proper moment although the unfortunate victim had nothing to do with gangster, automobile driver, airplane pilot or careless workman or housemaid. He was attending to his own business and was innocent of any offense. Such casualties are so frequent that we have little interest in the fate of the individual victim. We are more interested in the curious and unusual manner in which Fate operates.

But many a man contracts disease or meets death in such subtle ways that he is not generally recognized as an innocent bystander. Only physicians and a few wise laymen know his fate for what it really is—an unfortunate and fatal accident by a means no less deadly because it

*Given over Radio Station WGN, October 31, 1933, for the Educational Committee, Illinois State Medical Society.

is invisible. The organ of Fate in this instance is neither gun nor automobile carelessly handled, but disease germs disseminated by some ignorant or careless carrier.

Let me give you a few examples. Your child, in full health because you have given adequate care to his diet, exercise and rest, goes to school. Another child in the very early stages of scarlet fever or with a very mild form of the disease, is in the same class room. In some way he unintentionally transmits the germs to your healthy child and you have the red card of quarantine on your door for six weeks and possibly a visit from the undertaker. Your child may acquire diphtheria in the same way. He is an innocent bystander whose fate is only less spectacular than that of the victim of a stray bullet.

A few years ago a group of distinguished people attended a banquet and, among other things, were served raw oysters. Two or three weeks later several of them were in bed with typhoid fever and some of them died. It was subsequently learned that these oysters had come from waters that had been carelessly contaminated by city sewage. There were fatal cases of typhoid fever in Chicago at that time from the same sources. Let me hasten to add that the oyster dealers compelled that city to cease contaminating their waters with sewage and as a result of increased vigilance raw oysters are now a reasonably safe article of diet. But the unfortunate banqueters were in a very real sense, innocent bystanders.

Amebic dysentery is a tropical disease which is occurring with increasing frequency in Chicago and its vicinity. In the tropics this disease is usually acquired by drinking contaminated water. In temperate climates it is commonly transmitted to unsuspecting persons in food handled by carriers of the infection. A number of carriers have been discovered in Chicago. Amebic dysentery is usually a chronic and disabling disease. Sometimes it runs an acute and fatal course. It is characterized by diarrhea and pain and the passing of blood and mucus at intervals, all due to extensive ulceration of the lower bowel. About 25 per cent of the cases are complicated by a huge abscess in the liver.

The most pathetic of all innocent bystanders is the new-born baby rendered hopelessly blind by gonorrheal infection of the eyes during the process of birth. More than one-fith of all blind persons in the United States and more than three-fourths of all blind children under five years of age lost their sight from this preventable disease. They are the victims of ignorance and prejudice. For many years the medical profession has urged the passage of a law requiring the treatment of the eyes of *all* newly born infants, *immediately* after birth with a very dilute solution of silver nitrate. This is a harmless, but effective prophylactic procedure. With the support of the Illinois Medical Society and the Society for the Prevention of Blindness, such a law was recently passed by the legislature of this State. When its provisions are carried into effect the number of blind children under five years of age will be reduced by three-fourths, and the most pitiable of innocent bystanders will be saved from the total darkness of sightless eyes.

Dr. Woods Hutchinson many years ago declared that "germs can neither run, jump nor fly. They have to be carried." The carriers of disease germs, whether they be human or insect carriers, are generally ignorant of their state and quite innocent of intentional offense. This very fact renders them more dangerous. The carrier of whooping cough, like the far more deadly rattlesnake, makes his presence known. The human carriers of the germs of typhoid fever, or amebic and bacillary dysentery, of scarlet fever and diphtheria, of cholera, and the insect carriers of bubonic plague and malaria, approach insidiously and strike in the dark.

The people of a city whose water supply is contaminated by its own sewage, as was that of Chicago before the digging of the drainage canal, can expect, and as a fact do have, a high death rate from waterborne infections like typhoid fever, the dysenteries and cholera. The citizens of such cities are like soldiers in battle. They are constantly exposed to danger and death. But under modern sanitary conditions the general population is well protected against wholesale infection. It is under just these conditions that the individual carrier of disease becomes a special menace because of his ignorance and insidiousness and because he is not suspected. His victims are the innocent bystanders of disease transmission.

There is a significant difference between the innocent bystanders who succumb to disease and those who are the victims of the strange and

bizarre accidents recorded in the daily papers. Little can be done to protect the helpless pedestrian against wild bullets and wilder drivers of cars. But much can be done to protect the equally helpless prospective victims of carriers of disease germs. Vaccination against smallpox has been proved such an efficient means of protection against that disfiguring disease, that it should be voluntarily utilized by every intelligent person. Better still, universal vaccination should be required by law, in order to protect the general population against the potential danger from the ignorant, the careless and the selfish who will not submit voluntarily to this practically harmless measure of defense. An almost equally complete protection can now be obtained against typhoid fever, scarlet fever and diphtheria.

In calling attention to the insidious danger to your health from carriers of the germs of disease, it is not my intention to arouse unnecessary fear or to make you suspicious of your new cook or your neighbor. In pointing out the potential danger I have also indicated the means of avoiding it. Seek the counsel of your family physician as to the advisability of acquiring protective immunity for yourself and your children against diseases communicated by carriers of infection. Don't be an innocent bystander in matters of health.

COMPLICATED FRACTURES OF BOTH BONES OF THE LEG AND THEIR TREATMENT*

E. B. MONTGOMERY, M. D., F. A. C. S.
QUINCY, ILL.

My excuse for presenting another paper on such a hackneyed subject as fractures, is their increasing importance as a result of the hurrying conditions of modern life, particularly the speeding automobile.

This has been recognized by reports of Fracture Committees of our various medical organizations and the fracture exhibits made at the meetings of the American Medical Association and the American College of Surgeons.

My reason for selecting fractures of both bones of the leg as my subject is my personal experience with this type of fracture, as well as the difficulties others have found in their treatment.

*Read before the Section on Surgery of the Illinois State Medical Society, Peoria, May 17, 1933.

The late lamented Sir Robert Jones, in one of his lectures said, "If I were asked which fracture was the most difficult to reduce in the lower limb, I should say fracture of the tibia and fibula in the lower third." I might add, as a result of a personal experience "and to keep them reduced."

For in the long period of professional practice, the only malpractice suit with which I was ever threatened, was due to a fracture of this kind in which reduction was properly made and in which later muscular contraction reproduced over-riding in an oblique fracture of the tibia and fibula at the junction of the middle with the lower third. The use of ordinary splints or of Plaster of Paris will not retain the bones in reduction and some form of continuous extension and counterextension becomes necessary.

When, in addition to a fracture of this kind, you have a comminution of both bones and extensive injury to the soft parts, the problem becomes a still more difficult one. In such cases, skeletal traction and the Thomas splint is recommended by Scudder, Wilson, Cochrane, Kellogg, and Speed in their books on fractures and Eliason in his article on these cases in Nelson's Loose Leaf Surgery, falls in line with the other authorities. Hitzroth, one of the editors of "Oxford Loose Leaf Surgery," in a paper on "Fractures of Both Bones of the Leg," read before the surgical section of the A. M. A. in 1928, published in the "Journal" for January 26, 1929, says, "Fractures in which the prognosis will be poor, even with the best treatment, are the bad compound fractures with injuries by direct violence, fractures in elderly debilitated persons, fractures with nerve and vacular injuries."

These fractures, he thought, required especially skilled and trained treatment and in the discussion of his paper, the only method suggested by any of the discussants were: *First,* extension by adhesive tape and weight traction; *second,* skeletal traction by tongs in the malleoli of the tibia, the leg suspended in Thomas splint; *third,* primary operative treatment and plating of the bones.

In the February, 1933, number of "Surgery, Gynecology and Obstetrics" in an article entitled, "Skeletal Traction with the Steinman Pin. Results Obtained in 52 Cases of Fracture of Both Bones of the Leg" by Drs. Lindsay and McKlown, a report is made of these cases occur-

ring in the Orthopedic Section of the Department of Surgery of the Yale University School of Medicine. These cases are admirably reported in detail and the technique used, well described.

After incision of the soft parts overlying the os calcis, the Steinman pin is passed through the bone by hand drill and brace. Traction is made by calipers on the projecting ends of the pin, the leg being suspended in a Thomas splint. After four to six weeks, the pin is removed and the leg is put up in a Plaster of Paris boot. This is removed in four to six weeks and the leg is then kept in a Delbet removable splint for another four weeks.

While this is probably one of the best forms of skeletal traction for use in these cases, the purpose of this paper is to present a fourth alternative which seems to me, as a result of considerable personal experience, (not nearly as many as 52 cases) to be more desirable than the other methods outlined.

I refer to the use of the Hackenbruch "Distraction clamp," which I here show you and the use of which is shown to better advantage demonstrated by the lantern slides. While little or no mention is made of this method in the text-books, or in current English medical literature, it may seem presumptuous for me, on the basis of comparatively few cases, to recommend this method. A few cases well studied, however, frequently prove more illuminating than many not so well analysed. Moreover, there is abundant confirmation of my experience in current German medical literature and Hackenbruch has written a book of about 200 pages on the subject, a part of which I shall take the liberty to present to you in translation.

While the use of the clamp, as he presents it, has been highly satisfactory in especially refractory cases coming late under observation, in which reduction might prove difficult, from too great pressure on the soft parts, the use of the Steinman pin passed through the os calcis may prove a useful adjunct.

Momburg, who has had the most extensive use of this combination, reports it as highly satisfactory and Kirschner has made use of lubricated chromium plated piano wire, passed through a hollow needle of suitable caliber around the os calcis. Traction on the projection wire being used temporarily to assist the clamp and being removed in a few days.

In all complicated compound fractures, it goes without saying that loose fragments of bone and injured tissues must be surgically removed and the continuous use of the hychlorite method of disinfection used. This can readily be done with the Hackenbruch clamp as large windows completely surrounding the leg, permit complete inspection and painless handling of the wounds.

The patient is enabled to be up and about on crutches even in severe injuries in a comparatively short time, which contributes greatly to the rapid improvement of the general health and, in consequence, more rapid healing of wounds.

A brief description of this clamp, along with some lantern slides may serve to supplement a view of this clamp which I am sending about for your inspection. It is a ball and socket jointed splint made of chromium steel with great exactness to insure against breakage. It is assembled out of three individual parts. *First,* a screw rod with the screw grooves running both right and left, which in its center has a headlike enlargement. This is pierced by four bored openings which enables it to be easily turned by a steel pencil inserted in them. *Second,* a plate at either end with a ball and socket joint which is easily adjusted to any required position. If these clamps are embedded in Plaster of Paris, applied to the leg and the plaster then divided circularly at the level of the fractures, the two halves of the splint, by a simultaneous turning of the screw rods of both sides, may be made to separate two and one-half to three inches, thus providing abundant extension and counterextension, which is permanently retained. The possible changes in direction, which may be made by the ball and socket joints, in getting a correct position and line of fracture, is best shown by these lantern slide pictures.

These are all made possible by opening the ball and socket joint of the clamp, making such alterations as are found necessary or desirable and then fix the joints in the position attended by the set screws provided.

The presentation of the history of one of the severe cases seen by me may serve as typical of the course followed in all of them.

Mrs. D. Z., aged 70 years, on December 12, 1928, while crossing the street, was struck by a rapidly moving auto, her left leg receiving most of the impact, which produced a compound comminuted fracture of both tibia and fibula at the junction of the middle and

lower third of the leg. Fragments of the tibia protruded through two openings on the inner side of the leg and there was considerable bleeding from branches of the anterior tibial artery, this continuing for several days after the accident.

After preliminary cleansing of the leg and removal of some fragments of bone, the leg was placed in a posterior leg splint of aluminum and dressings changed frequently. On the fourth day, a gangrenous area, the size of an orange, appeared on the posterior aspect of the leg. As there was considerable shortening and overlapping of the bones, the fracture clamps were used, the method of use as advised by Hackenbruch being followed.

The leg was flexed at right angles at the knees and the thigh similarly flexed at hip joint and by traction on femur and counter-traction on foot and ankle, the reduction was made as far as possible. After reposition of the fragments, the limb being held in the same position, pads of absorbent cotton were bandaged in places about the head of the tibia, above, below and about the ankle and fixed in place by bandages, sterile gauze was placed over the wounds and held in place.

Then from the knee to the upper part of the foot, a broad strip of cotton was placed for protection on the crest of the tibia and the entire leg carefully bandaged from the toes to the patella, so that the gauze covered part was exposed and could be freed between the bandage layers. Then thickly over the ankle was bound a strip of cotton about two inches in breadth. This served to protect the skin when the plaster bandages were applied. Then the plaster bandages were applied from the toes to the patella, incorporating two Gigli saws, front and back, at the level of the fracture. The bandages were strengthened by laying two or three strips of plaster bandage lengthwise.

As the plaster hardened a cut divided it front and back with the Gigli saws incorporated in the plaster. Then the clamps were bandaged in place above, below the knees and below, above the ankle on either side and fixed in place. After the plaster, fixing the clamps, was hardened, it was separated, as shown in slides, by the Key.

A still larger window was cut in the cast posteriorly so that the sloughing portion of the leg might be dressed at intervals with a diluted alcohol dressing. A very considerable portion of tissue, overlying the gastrocnemius muscle, separated after five weeks and four weeks later skin grafts were placed over the extended raw surface and closed the gap in a very satisfactory manner. Bony union, without shortening or any noticeable deformity, occurred, and five and a half months after the receipt of the injury, she walked without cane or crutch and two months later without a limp. The x-ray pictcres here shown, give some idea of the severity of the fracture and the final result. Other severe cases, which might be detailed, had a like satisfactory outcome.

The cases which I have treated were of this complicated character, so severe that they could not be treated in recumbency and operative treatment offered still less promise.

One advantage of this splint is that one can always check up by x-rays on position and make adjustment under fluoroscope, and hold the position without removing splint or greatly disturbing the patient.

In conclusion, *first,* in complicated fractures of tibia and fibula of the lower third, we have a fracture difficult to reduce and still more difficult to hold in reduction. *Second,* the methods in general use are forms of skeletal traction, requiring bed confinement and making more difficult the treatment of complications. *Third,* the method here described, is effective in the severest types of cases in retaining the bones in any position desired and does not necessitate long bed confinement, the general health of the patient being correspondingly improved.

DISCUSSION

Dr. J. H. Bacon, Peoria, Ill. The surgeon who is treating one of these compound comminuted fractures produced by a high powered machine, automobile or otherwise, is often worried. Lovett has wisely said, concerning these fracture cases, "ask yourself what are you trying to do and are you doing it." It is one thing to get the fragments of bone in position and it is quite another to hold them there. Any method that will give good results is a method that we are looking for. It is not always one method that will suit every case. Every comminuted fracture is an individual case of itself and must be treated individually. We often neglect to care for the soft tissues properly and many of the bad results are not from malposition but from malnutrition.

There might be added another method which we often hesitate to select, and that is amputation of the leg itself. This is sometimes necessary on account of the financial status of the individual, because in times like these many cannot have the financial backing which is necessary for a long treatment. I can never forget a case I saw in Massachusetts, in which a small boy of eight had been brought into the Peter Bent Brigham Hospital suffering from extensive injuries to a leg brought about by being caught in a wagon wheel. There were multiple fractures of the leg and thigh, and multiple contusions of the soft parts. Two eminent surgeons saw the case. One said, "amputate the leg," and the other, "I think I can save the leg." The first one replied, "it is your job." I saw the end result after nine years when this boy of 17 was in a state institution where he had been during this long period. He had had, as I remember, somewhere around twenty different operations. He had a leg on which he could barely stand but he could not get about in cold weather. Sometimes the end results not in mortality but in morbidity require amputation. We should think of it ser-

iously in many of these severe cases, especially if there is much mangling of the soft parts and much damage to the blood supply.

There one possible objection to the use of this instrument shown by Dr. Montgomery. Are you interfering too much with the blood supply when you put on pressure traction on the distal part of this leg? It boils itself down into one's ability to use the apparatus and the ability to properly apply plaster.

Another sidelight on the use of plaster. We all like to see a well applied cast and the fine appearance that plaster makes, but are we sufficiently concerned with the part of the plaster that is coming into contact with the individual himself. Are there any wrinkles there? It does not matter if there are wrinkles on the outside of the cast but it does make a big difference if there are any on the inside of the plaster, as their presence may seriously interfere with the blood supply of the tissues.

This has been a very interesting though now a new method. It is one that has given results in Dr. Montgomery's hands. Any method or apparatus that gives results should be considered. This apparatus has the advantage of being small and not expensive, and of being easily applied.

Dr. E. B. Montgomery, Quincy (Closing the discussion): I presented this only as a method that was successful in my hands in lessening the period of confinement to bed and in the hospital, which are important features especially in these days.

THE MEDICAL MANAGEMENT OF HEPATIC DISEASE*

CHARLES A. ELLIOTT, M. D.
Professor of Medicine, Northwestern University Medical School
CHICAGO

Physiologic and chemical studies on experimental animals have provided a clearer understanding of liver function and have made possible the formulation of general principles of treatment. Clinical application of these principles has, however, lagged despite the appearance in the literature of numerous contributions on the subject. As a result the average clinician has neither accepted nor followed a definite line of attack in the general management of hepatic disease comparable, for example, to that almost universally adopted in the treatment of cardiac disease.

The liver is an organ of exceptional interest. Originally a simple tubular gland whose chief relationship was with bile capillaries, it has, in the higher forms of life, become especially concerned with matters of metabolism and nutrition, and its parenchymal cells have acquired an intimate contact with the blood system. The seeming simplicity of the undifferentiated liver cells is in reality deceptive. Their mass is constantly changing. The individual cells as well as the liver as a whole are constantly changing in unison with physiologic and pathologic processes; they are peculiarly sensitive to physical, chemical and biologic reactions. A particular feature indicating the apparently primitive nature of the liver parenchyma is its extraordinary ability to repair damage and, by means of regeneration, to restore normal function in areas where destruction has occurred.

As a result of pathologic change a number of characteristic events result. The liver is not liable to the pathologic conditions usual in other organs. Ordinary acute inflammation, for example, is uncommon; tuberculosis is rare except as a part of a generalized condition. The peculiar things that happen in the liver are four in number, and represent a fairly constant sequence of events. First, *parenchymatous degeneration:* This results from a variety of influences such as infections, toxemia from infection, chemical poisons, and anoxemia. Cloudy swelling is produced which may be transitory, or the process may go on to fatty degeneration or even to necrosis which may be focal or diffuse. Fatty degeneration is the term long applied to a condition of cell degeneration and of functional disturbance leading to increased deposition of fat. Serious damage to the liver cell leads to necrosis which may affect almost the whole mass of liver cells or be limited to groups of lobules, or of certain cells in the liver cell cords. Acute, diffuse necrosis (yellow fever) represents the reaction of highly sensitive cells to a toxic agent without injury to the interstitial cells or the framework of the liver. Since the liver cells form the substantial, if not the only elements of the wall of the bile capillaries, jaundice is inevitable. Both severe and lesser degrees of damage which may result in recovery because of repair or of regeneration occur in yellow fever and appear to be present in many cases of catarrhal jaundice. The term "acute hepatitis" has been applied to many cases of milder liver damage resulting from various causes. In ordinary cirrhosis the process is gradual, slow and progres-

*Abstract of a Clinical Lecture, illustrated with lantern slides, given before the Section on Medicine, Illinois State Medical Society, at Peoria, on May 18, 1933.

sive. Second, *regeneration:* Regeneration of liver cells unharmed or only slightly damaged in areas of necrosis is striking, rapid and inevitable if time permits and conditions are favorable. It has been estimated that as much as 150 grams of active liver cells may regenerate in one day and that it is possible for six-sevenths of the liver to regenerate in the course of six weeks. Regeneration will not occur during starvation or when the common duct remains obstructed. A high carbohydrate intake favors rapid regeneration. Third, *fibrosis:* Connective tissue condensation occurs when the volume of liver cells is reduced by acute necrosis without injury to interstitial tissue and restitution to anatomical integrity may occur without cirrhosis. Liver injury affecting the connective tissue as well as the liver cells, as in true acute inflammatory lesions and in the chronic progressive damage of portal cirrhosis, produces marked increase in connective tissue and subsequent contraction. Fourth, *portal stasis* occurs when the fibrosis prevents adequate circulation of the portal blood through the liver. Other venous channels attempt to compensate for this defect and may succeed indefinitely without the appearance of symptoms. With decompensation ascites appears.

Of chief importance in treatment is recognition of known facts in regard to the role of the liver in carbohydrate metabolism. The liver is constantly changing during the process of food storage. It stores exogenous carbohydrates as glycogen apparently with great facility. The liver cells are concerned with the conversion of protein (as much as 58 per cent.) and fat (estimated at 10 per cent.) into carbohydrate, but apparently perform this function with greater difficulty. During fasting the glycogen content of the liver is greatly reduced; the cells appear shrunken and the bile capillaries and especially the blood capillaries appear relatively large. By the administration of glucose one can produce an increase in the size of the liver cells and, in fact, of the entire liver mass. A low glycogen content prevents normal efficiency of function. Conversely, a liver well stored with glycogen increases resistance to damage. The administration of glucose has been shown to favor regeneration and to decrease the coagulation time of the blood in cases of experimental liver injury. For these reasons a high carbohydrate intake is of first importance in liver disease as a matter of prevention and as a matter of treatment both of acute and chronic processes. The average normal diet contains from 400 to 500 grams of carbohydrate. At least this amount, and more if at all possible, should be supplied in liver disease. If this amount can be taken by mouth or be given by means of a stomach or duodenal tube in the form of carbohydrate foods, including fruit juices and glucose, no other route of administration need be used. It may be necessary to resort to the use of intravenous glucose (10 per cent. administered slowly). Rectal administration is not well borne and is relatively worthless. In acute cases of liver damage no attention need be paid to the amount of protein permitted provided it is low in order to spare the overburdened liver cells and prevent additional toxemia. In chronic cases the protein intake should be sufficient to meet the requirements of the individual (about 1 gram per kgm.). The fat allowance should be low, particularly in cases of jaundice during which clinical experience indicates fat is poorly tolerated.

In cases of obstructive jaundice the administration of whole bile is indicated on physiologic grounds. In experimental studies bile has been found necessary to keep animals in good condition; in man the need is less apparent. Bile from a drainage tube may be reintroduced or ox-bile may be used.

In liver disease associated with portal stasis and decompensation (ascites) the newer diuretic regimen recommended in cardiac and renal edema has frequently proved of value in ridding the patient from and keeping him relatively free of ascites. Briefly, the elimination of sodium chloride from the diet, the use of large amounts (9 to 10 grams daily) of ammonium nitrate or chloride and the repeated use as required of a mercurial diuretic such as salyrgan, employing the evidence of ascitic accumulation as well as weight and water balance charts as a guide, will obviate the necessity of paracentesis and keep the patient in relatively good condition. The danger of hematemesis, however, remains. Obviously, this form of treatment can not be expected to be of value if serious loss of hepatic function is present.

THE EFFECTS OF AQUEOUS SOLUTIONS ON THE NASAL CILIATED EPITHELIUM*

IRVING F. BARNETT, M. D.
CHICAGO

This subject is not one of recent discussion as Piorry in 1844 as quoted by Wright [1] stated that water was injurious to the upper respiratory tract. However, as accepted authorities in recently written text-books vary in their statements from interdiction of all aqueous solutions in the nose [2] to use of irrigations by aqueous solutions as detergents and stimulants to the muscle fibre of the swell body of the turbinate, this [4] condition and the recognition of extreme importance of the nasal mucosa as a defensive mechanism against bacterial invasion makes it important to decide this mooted problem.

The defensive mechanism of the nasal mucosa can be divided into a [5]

A. Major and interdependent trio.
   1. Mucus secretion.
   2. Ciliated epithelium.
   3. Phayocytosis and anything interfering with ciliary activities may cause stagnation with resulting infection.[6]

B. Minor: Local immunity, reaction and lysozyme activities.

[7] The ciliated epithelium lining the posterior two-thirds of the nose, extending into sinuses by its rhythmic motion in the mucus not only drains downward to nasal floor and posteriorly to the pharynx, actively draining the nasal cavities but inactively by the tenacity of this same mucus the nonciliated anterior one-third of the nose.[8] The inhibitory action on bacteria of the mucus is attributed to its motility and chemistry of its destruction by bacteria. Therefore the persistence of ciliary activity is of prime importance.

Regeneration of ciliated epithelium has recently been proven, notwithstanding metaplasis is the rule when continued destruction occurs.

Aqueous solutions can act deleteriously on the thermally, electrolyticly and generally very sensitive ciliated cells in either direct or indirect manner.

Indirectly by solution of the mucus with exposure of the cilia to the air with resultant destruction. This unfortunately is the action of the detergent solutions as borax, etc.

Directly the extent,[9] effectiveness and direction of ciliary propulsion are greatly modified by excess of moisture which creates gravity currents flowing over neutralizing ciliary currents. These currents are determined by the amount and viscosity of the fluid and also by any one or combination of the following which vary directly with the quantity, frequency of medicant in solution, strength of solution and traumatic method of use, viz:

1. Solutions [10] of sodium chloride in 0.9% to 2% showed an inflammatory reaction directly proportionate to the above both macroscopically and microscopically with sinus filled with fluid in those examined within 25 minutes after irrigation.

2. Trauma by force of fluid tears of the less strongly cemented ciliated cells.

3. Thermally [11] ciliated epithelium is especially sensitive to cold.

4. Strength of solution [12]—
   a high sensitivity to osmotic pressure another characteristic of ciliated epithelium.

5. Electrolytes effect principally the liquefaction of the cilia by effecting cell membrane and surface tension of the ciliated cell as by pure sodium chloride [13] also the proportions of calciumons on the presence of which is dependent ciliary activity.[14] Practical application is by the combination of preferably magnesium with sodium chloride which is antitoxic both to liquefying and calcium precipitating effects.

6. Strength of solutions producing effects by other than osmotic, detergent or electrolytic which are common ingredients in nasal solutions as menthol, eucalytol and camphor produce deleterious results varying with the strength of solution 1-5% and in the manner listed.

Conclusions—

1. Aqueous solutions on ciliated epithelium in the nose should be used circumspectly.

2. Large quantities, over a period of time, especially when forcibly applied, are contraindicated.

3. An isotonic amphoteric antitoxic solution at body temperature non-irritant solution for a short period of time is the best.

*Read before Section on Eye, Ear, Nose and Throat, Illinois State Medical Society, Peoria, May 17, 1933.

4. Colloid silver salt solutions are the least harmful and effect results by ionization and catharsis of glands.

REFERENCES

1. Stark, W. Berkley: Irrigation aqueous solution and effect on the membrane of the upper respiratory tract. Arch. Otolar. 1928. Volume 18, Page 1.
2. Pratt, F. A.: Internasal Surgery. F. A. Davis Co., Philadelphia. 72. 1924.
3. Ballinger: Diseases of the Nose, Throat and Ear. Lea & Fibiger, Philadelphia. Page 459. 1930.
4. Thompson, Sir St. Clair. Castle & Co., Ltd., London. Page 57. 1926.
5. Hilding, Anderson: Drainage of Nasal Mucus. Arch. Otolar., Vol. 15, No. 1. Jan., 1932.
6. Dean, L. W.: Etiological Effects in Nasal Sinus Diseases. Trans. Am. Laryn. Rhin., Otolar.
7, 8. Hilding, Anderson: Drainage of Nasal Mucus. Arch. Otolar. Vol. 15, No. 1. Jan., 1932.
9, 10, 11. Proetz, Arthur W.: Physiology, Annals Otolar. Page 970, Rhin. & Laryn. Vol. 38, No. 4, Year 1929.
12. Osterhaut, W. J.: Injury, Recovery and Death Relative to Conductivity and Permeability. J. B. Lippincott & Co., Phil. 1922.
13. Wenner, W. F., and Nemous, P. R.: The Mucosa of the Maxillary Sinus of the Rabbit. Annals of Otol. Vol. 15, No. 2, 1932.
14. Ballinger: Diseases of the Nose, Throat and Ear. Lea & Febiger. 1930.

THE SURGICAL TREATMENT OF SPONTANEOUS AND TRAUMATIC DETACHMENT OF THE RETINA*

C. F. YERGER, M. D.
CHICAGO

Theories of the origin of detachment of the retina go back to the year 1818 when Wardsop both by clinical and pathological observations thought that it was due to a serous exudate from the choroid. In 1870, DeWecker was the first to point out the frequency of retinal tears and their pathological importance in detachment of the retina. Leber, in 1882, was the first to suspect that the primary lesion was in the vitreous. Leber, Vogt, Gonin, Lindner, Elschnig and others believe the cause of a detachment of the retina is the hole or tear in the retina.

Gonin's contribution to the pathogenesis of detachment of the retina and its application to the surgical treatment has stimulated much discussion and research. He believes that primarily there is a pathologic change in the vitreous, i.e., a partial liquefaction occurs, as is frequently seen, as fine floating opacities in senile and myopic eyes. A partial detachment and retraction of the vitreous follows, as a result of the vitreous separating into a non-albuminous watery fluid and a vitreous gel portion. Subsequently, atrophic and degenerative changes occur in the retina near the ora serrata, since here it is the thinnest with the exception of the fovea and lacks the support of the optic nerve fibers and retinal blood vessels and is more vulnerable to pathologic changes as, senile cystic degeneration and chorio-retinal atrophy, consequently, it is here that holes and tears, in the retina are most common. Any abrupt movement of the eye or body or a contusion of the eye could cause a hole or tear to result in the atrophic and degenerated retina if the surrounding area was supported by a fluid vitreous. The non-albuminous watery fluid of the vitreous percolates through the hole or tear in the retina, becoming the subretinal fluid which detaches the rod and cone layer from the underlying pigment epithelium. Anderson points out that the invagination of the primary optic vesicle leads to the formation of two layers which later come into contact but do not unite. It is this contact line which appears to be a developmental weak spot that becomes the line of cleavage in retinal detachment. As the subretinal fluid increases in amount, the detachment increases in size. Both the pre-retinal and the sub-retinal spaces are filled more or less with this watery fluid which is a transudate and not an exudate due to inflammation. The vitreous gel retracts to the region between the equator and the ora serrata and usually shows extensive adhesions to the retina. The retina yields to jarring and a hole or tear results at the ora serrata or where the vitreous bands form adhesions to the retina. The outflow of the fluid vitreous through the hole or tear causes and maintains the detachment.

1. *Gonin's Operation.* Gonin believes that a detachment of the retina can take place only, if a hole or tear of the retina is present and reasoned that if the hole or tear was the fundamental cause of the detachment, the early evacuation of the subretinal fluid and closure of the hole or tear should result in a cure of the detachment. This he accomplishes by igni-puncture, using the Pacquelin cautery over the site of the hole or tear and causing an agglutination of the retina and choroid and obliteration of the hole or tear. This demands that an accurate external localization of the hole or tear must be made.

*Read before Section on Eye, Ear, Nose and Throat, Illinois State Medical Society, Peoria. May 17, 1933.

For this purpose, the globe is divided into meridians corresponding to a clock dial. The pupil must be widely dilated. Ascertain the meridian upon which the hole or tear is located and the distance it is from the ora serrata by estimating the number of disc diameters it is situated from the extreme periphery of the visible fundus. We can now compute the distance the hole or tear is located from the limbus. The diameter of the disc is 1.5 m.m. and the distance of the ora serrata to the limbus is 8 m.m., therefore if we found the hole or tear located at 2 disc diameters from the extreme periphery of the visible fundus, the hole or tear would be located 11 m.m. from the limbus. The involved meridian being ascertained, it is indicated by two India ink dots on the limbal conjunctiva of the clock dial. Pass a knotted black silk thread through the further dot of ink from the hole or tear and then through the other dot of ink. Measure the distance of the hole or tear from the limbus along the meridian involved and indicate the location of the hole or tear with an alcoholic solution of gentian violet, where the cautery is to enter the globe. The meridian alignment should parallel these three points. A conjunctival flap is made over the area and a scleral trephine done over the hole or tear. A Graefe knife enters the trephine opening to pierce the choroid to evacuate the subretinal fluid, which allows the retina to fall back to its former place next to the choroid. The glowing cautery is now applied where the knife was withdrawn. The heat cauterizes the retina, choroid and sclera, forming a cicatrix which will close the hole or tear in two weeks. The essential points of Gonin's operation are: the finding of the hole or tear, its accurate localization on the globe, the evacuation of the subretinal fluid and the obliteration of the hole or tear by igni-puncture.

The contra-indications for Gonin's operations are: the inability to localize or the doubtful localization of the hole or tear, poor transparency of the anterior segment, extensive size of the tear, multiplicity of tears, location of tear at or near the posterior pole (22 m.m. from limbus is the limit), advanced age or poor general condition of the patient and the stabilization of the detachment with retention of fair vision.

Holes and tears in the retina were found in 77% to 100% of the cases of detachment of the retina. The location of the tears are in the region of the equator or anterior to it, 66% were at the ora serrata. One or more defects were most frequently found in the upper or lower temporal quadrant. Holes and tears are rarely found posterior to the equator.

Seventy-five per cent. of the detachments of the retina were located in the temporal hemisphere of which 65% were in the inferior temporal quadrant and 10% in the superior temporal quadrant; 86% were in the inferior hemisphere of which 65% were in the inferior temporal quadrant and 21% in the nasal quadrant.

2. *Larsson's Diathermy Method.* The conjunctiva is stripped from the corneo-scleral margin over the area of detachment. The active electrode is applied with a weak current for 5 seconds to the sclera 8-9 m.m. from the limbus to avoid the ciliary body and lens. An opaque dark colored zone is produced and the sclera flattened out. One or two Elliott trephine openings are made in the sclera of the treated area and with a Graefe knife incise the exposed choroid and the subretinal fluid removed. The patient is kept in bed for 14 days with both eyes bandaged. Atropine is used while the eye is red. Larsson states that immediately after the operation, the retina is detached, but in favorable cases it becomes reattached in from 5 to 8 days. The reattachment occurs by producing an adhesive choroido-retinitis and lessening the secretion of the chorio-capillaris due to atrophy of the cicatrized choroid.

3. *Meller's Operation.* This is a modification of Larsson's operation, by preceding the application of the active electrode by 2 to 6 scleral trephines in the area of the detachment and in at least one of the trephine openings, the subretinal fluid is evacuated. A 3 m.m. active electrode is applied until a dry brown surface is produced. A binocular bandage is applied and the patient remains in bed for 2 to 3 weeks. The cauterized area of detachment shows chorio-retinitic atrophy with pigment formation. The early reaction is frequently a cystic detachment of the retina, even where no detachment previously existed, which later disappears. The rationale of the operation is to produce widespread adhesions between the retina and the choroid.

4. *The Guist Operation.* The conjunctiva is

anesthetized with the instillation of a 2% solution of Holocain and to favor the maximum rotation of the globe, the recti muscles in the field of operation are injected with 2% Procain HCL solution. The incision is made long and parallel with the limbus and over the insertion of a rectus muscle. The globe is rotated to give the necessary exposure of the sclera over the area of the detachment. The scleral trephines are made with 0.5 m.m. trephine, the number depending on the extent of the detachment. The scleral perforations should not include the choroid otherwise the vitreous will prolapse. The trephined scleral areas are excised exposing the choroid which is treated with potassium hydroxide pencils which are applied dry to the choroid for two or three seconds and then neutralized with 0.5% acetic acid and then irrigated with normal salt solution. Before beginning the chemical cauterization, see that the field of operation and the caustic potash point are perfectly dry. The number of trephine openings vary according to the case, from 5 to 30. The subretinal fluid can be drained by perforating the choroid through one or more of the treated scleral trephine openings with a punctum dilator. If the rectus muscle was cut at its insertion and a double armed silk suture placed in the stump, the muscle is now reattached and the conjunctiva sutured. A binocular dressing is applied and the patient kept in bed for several days and after 8 days is given the use of eyes, using the small aperture glasses.

According to Lindner, the Guist operation does not cause foreshortening of the retina, with resulting new tears and recurrences and does not require exact localization of the holes or tears. It does not seriously injure the retina as does the Pacquelin cautery, and choroidal hemorrhage is rare. Its main disadvantage is, it is time consuming, taking from 1½ to 2½ hours. There is also the danger of fistula formation and of not neutralizing the potassium hydrate and acetic acid and the entering of these chemicals into the vitreous, of producing an edema of the cornea and hemorrhage of the choroid. To reduce the liability of this, use a small blunt pointed probe instead of a knife. If one punctures the choroid, in making the scleral trephine, the eye may become soft, due to the premature drainage of the subretinal fluid or prolapse of the vitreous and then it may become difficult or impossible to continue the operation.

We should now briefly consider the pros and cons of the above methods of operation for the cure of detachment of the retina. Gonin and his followers claim, it is only necessary to make a single and direct attack on the hole or tear in the retina and close it. Whether this procedure per se is insufficient as many claim and not as important as the production of extensive adhesions between the detached retina and choroid which would also include more or less of the hole or tear, is a question that the future must decide. Mayer has shown experimentally that the retinal hole or tear is not the only factor in maintaining a detachment of the retina.

Gonin insists that unless we accurately localize the hole or tear in the retina and permanently close it, the detachment will recur. Deutschmann does not believe that the retinal hole or tear is the primary cause because it frequently ceases spontaneously or after igni-puncture without healing the detachment. He believes the essential factor in igni-puncture is fixation of the relaxed retina to the choroid. DeWecker, Leber, Vogt, Gonin, Elschnig, Lindner and others believe, the retinal hole or tear is the primary cause of the detachment and that closure of the hole or tear is the prime factor while Deutschmann, Larsson, Meller, Guist, Linder and others take the view that it is not sufficient to treat the retinal hole or tear alone, but that the whole area of the detachment should form a complete adhesion between the retina and choroid. Of the four operations here described, two depend on diathermy (Larsson and Meller), one on igni-puncture (Gonin), and one on chemical cauterization (Guist). The electro-thermo-cautery should be substituted for the Pacquelin cautery because of the lesser liability of intra-ocular hemorrhage, and the production of new tears. Which one of these methods is most efficient and least injurious to the eye, the future shall determine. It is singular, that regardless of the methods used, the successful reports claimed for each is about the same, i.e., the results reported by Larsson, Meller, Guist, Lindner, Deutschmann and others are approximately as good as those claimed by Gonin who claims about 50% of cures, but others who have used his method could not obtain more than 15% in their com-

bined cases of cures and improvements. Guist and Lindner were dissatisfied with Gonin's method because they believed that it is not sufficient to treat the retinal hole or tear alone but that the whole area of the detachment including the hole or tear should be treated. The Gonin operation cannot be used in cases where it is impossible to find the hole or tear. It is difficult to locate the hole or tear and if one does not strike the hole or tear, one cannot expect a good result.

The question of how long the patient should remain in bed is an important one, but in these operations, there seems to be no unanimity and varies from a few days (Guist), to 28 days (Larsson) and the duration of the binocular compression bandage, from 8 days (Guist), to 14 to 21 days (Meller).

The results of Guist, Lindner, Larsson and Meller show that it is not necessary to localize the holes and tears, but rather to make an accurate external localization of the extent of the retinal detachment and attempt to cause an obliteration of the subretinal space with its holes and tears. The success of any of these operations depends upon the proper selection of cases, the accuracy of the technic, and the co-operation of the patient.

The prognosis depends to a large extent on the duration of the detachment; other factors being equal, the shorter the duration, the more favorable the outlook. From this standpoint, Gonin divides his cases into three clinical groups: 1, during the first three weeks; 2, from three weeks to three months, and 3, from three months to a year. His original teaching was that one should operate in three or four days after the onset. The amount of visual recovery depends upon the degree of degenerative changes that have occurred in the retina, the causative factor, the extent of the detachment and the presence of multiple holes and tears. Elschnig states that a retinal detachment is not cured when the retina becomes reattached; that vision is rarely restored even after a complete reattachment of the retina and a progressive loss of vision results. This brings up the question of what we should consider a "cure." The patient will judge the operative result solely upon the effect on his central visual acuity, while the surgeon may consider the ophthalmoscopic appearance of the detachment or the result of the perimetric examination more important. The visual acuity, peripheral fields and the ophthalmoscopic appearance of the detachment should be recorded at frequent intervals during the period of observation. We should not consider a case of reattachment of the retina as "cured" unless no recurrence has occurred after a period of at least three years. The results of treatment should be classified as, 1, cured; 2, improved; 3, no improvement, and 4, worse. A good functional and anatomic result over a period of three years should be classified as "cured." Sourdille, who had unusually good operative results in 179 cases, after a period of ten years, was able to report but one cure.

Gonin, in 300 cases of detachment of the retina in each of which a retinal tear was found, localized and obliterated by igni-puncture, reports that in detachments less than three weeks old 66% were cured; between six weeks and three months old, 51%; and in more than three months, 37% were cured. One should not be discouraged by post-operative recurrence and in cases where the indications are still present for operative interference reoperation should be done, thus Gonin reports but 29% cured by a single operation, 15% by two operations, 7% by three operations, one case required four operations and two cases had six operations. Angler, using the Gonin method, operated on 40 cases of which 20 cases or 50% showed reattachment. In 75 cases at Moorfields using the Gonin technic, Shapland et al report 32% "cured" and 16% improved; recurrence occurred in 25% of the cases within 7 days to 8 months. Deutschmann, doing igni-puncture in 539 eyes, claims as good results as from the Gonin technic. Lindner reports good results in 50% of 27 cases using a modification of Guist's operation. Larsson, using his diathermy method, claims about 50% of cures in 50 cases. For comparison of the results of the operative and non-operative treatment of detachment of the retina with rest and general dehydration treatment, Darier gives the percentage of cures in the latter as 10%.

Poor operative results may be due to, the operation being too long deferred, the inability to obtain post-operative immobility of the patient, advanced age or poor general condition of the patient, large size of the tear, multiple tears,

extreme posterior location of the tear, in the aphakic group, Gonin did not obtain a successful result, predisposition to post-operative recurrence, too many operations, and the occurrence of operative complications as vitreous hemorrhage which occurs from 7 to 10 days after operation when the Pacquelin cautery was used, uveitis, traumatic cataract, and subretinal hemorrhage.

4458 Madison Street.

BIBLIOGRAPHY

1. Amsler, M.: Observations on Gonin's Operation, Arch. de Oftal. Hispano-Am., Dec., 1931.
2. Anderson, J. Ringland: The Causes and Treatment of Detachment of the Retina, 1931, Cambridge Press.
3. Anderson, J. Ringland: Avulsion at the Ora Serrata, Brit. J. Ophthol., Nov. 1932, Vol. 16, No. 2, F. 641-670.
4. Darier: Detachment of the Retina, La Clinique Ophthal., Jan., 1919.
5. Deutschmann, R.: Ignipuncture in Retinal Detachment, Ztsch. f. Augenh. Jan., 1931.
6. Elschnig, A.: Retinal Detachment, Arch. f. Augenh., June, 1930.
7. Gonin, J.: Thermo-Puncture on the Obliteration of Retinal Tears in 300 Cases. Ann d'Ocul., 168:689, Sept., 1931.
8. Larsson, S. Treatment of Retinal Detachment with Endothermy and Trephining, Acta-Ophth., 8:192:1930.
9. Larsson, S.: Electro-Endothermy in Detachment of the Retina, Arch. Ophth. May, 1932.
10. Lindner, K.: Detachment of the Retina, Arch. f. Ophth., 127:12:1931.
11. Lindner, K.: Contribution to the Origin and Treatment of Idiopathic and Traumatic Detachment of the Retina, Arch. f. Ophth., 127:177:1931.
12. Mayer, L. L.: Detachment of the Retina and Its Surgical Therapy, Arch. Ophth., April, 1932.
13. Mayer, L. L.: The Vitreous in Experimental Detachment of the Retina, Arch. Ophth., June, 1932.
14. McKeown, H. S.: Detachment of the Retina, Arch. Ophth. 9:1:1933.
15. Meller, J.: Production of Flat Adhesions between the Retina and Choroid by Endothermy, Ztschr. f. Augenh., Oct., 1931, 75, 207-216.
16. Ormond, A. W.: Spontaneous Detachment of the Retina, Brit. M. J., May 24, 1930.
17. Shapland, C. A.: Hundred Cases of Retinal Detachment Treated by Cautery Puncture, Tr. Ophth. Soc. U. Kingdom, April, 1930.
18. Shapland et al.: Simple Detachment of the Retina, 75 Cases Treated at Moorfield's by the Gonin Method. Brit. J. Ophth. May, 1931.

DISCUSSION

Dr. Louis Bothman, Chicago: I would like to ask Dr. Yerger about his personal experience with these various types of operation. We have tried in private practice and at the University of Chicago the various operative procedures, and our experience with the Gonin operation has not been very satisfactory. We have had several cases where the results in the beginning were good, but almost invariably the retina became detached later on. In one case in particular the retina remained in perfect position for about fifteen months, after which there was total detachment, and this is the story with most cases of ignipuncture. Our best results have been with the Guist Lindner operation and while I cannot give the exact percentage, I should say that about 30 per cent. gave a very satisfactory result for a long period of time. At the present time we are trying the diathermy operation but none of these cases were operated on more than six months ago. We do not know what the final result will be, but the early results are satisfactory. With the Guist Lindner operation about 30 per cent. have been satisfactory and the detachment has remained in place for periods of as long as a year and a half.

Dr. George Francis Suker, Chicago: I think the question of detachment of the retina is a very interesting one, and looking back over a short period of time I do not know that the final results obtained are much better than those in years gone by. The patients have everything to gain and nothing to lose, it is true, and this has to be considered. In my opinion, in some operations, particularly the Lindner (not Guist), and at times the Gonin, truthfully a Lindner operation, do cause in some instances a shrinkage of the globe. I have some such cases on record.

Of all the operations, I think the Lindner or diathermy are the most justifiable. So far as recurrence of the detachment is concerned the Lindner operation offers the best results, but any recurrence at any time is more or less fatal. I have one under observation at the present time. The first ten days after operation there was an almost perfect field of vision with normal vision where previously it was just fingers in the temporal area. Within ten days of operation there was a very large secondary detachment in the inferior quadrant of the retina. Patient was put back to bed and subretinal fluid drained; more or less reattachment followed, but finally it detached again. This is a type of cystic detachment which is apt to follow any operations, particularly of the Lindner type. I do not believe that it is absolutely necessary to actually touch or cauterize the hole in order to secure reattachment. As long as you have an area of active inflammatory reaction in choroid and sclera surrounding the hole a reattachment is likely to be obtained. Repeated operations seldom give any worth while end results. The earlier the operation the greater chances for a success. Detachments of very long standing yield very poor results—that is, you can obtain reattachments, but functional results are usually nil.

In using the trephine it has been my custom to trephine in such a way as to have the sclera attached in a little area of the trephine and then clip it off. It is not necessary to have the entire sclera removed from the choroid, so long as there is only a thin section of sclera allowing choroid to shine through. The application of caustic for several seconds is sufficient. If necessary you can scrape out the remaining portion of the sclera, but as long as you have a pinpoint opening through which the choroid shines you can cauterize just as efficiently as in a large hole.

I think it is obligatory to put these patients to bed and to apply stenopeic glasses to the eyes and keep them flat before you operate for about ten days, with sweats and salt free diet, not necessarily bandaging them. This will give you mechanical assistance as the tendency is for the retina to settle down. This regime

will allow you to study the case better and often locate holes which were previously hidden from view by the folds.

I think it is essential to have your patient wear a glass with a pinhole opening for sometime after a successful operation. This is as near a functional aid as you can give in keeping the eyes quiet and yet function without unduly disturbing the retina, as in this wise the ocular rotations are extremely limited.

Dr. Earl L. Vernon, Chicago: Dr. Yerger states that the best results were obtained when operation was performed early. The thing I have to decide, in many cases, is when did the detachment occur? Often, the patient gives the history of a recent trivial injury with resulting detachment, large or small, as the case may be. I have not been able to tell by looking at it, if the detachment is a month or six months old; of course, we know they tend to become total. Is there any way to determine the age of the detachment in these so-called early cases other than depending upon the history of the case?

If possible, I should like some discussion on the point.

Dr. C. F. Yerger, Chicago, (closing): Replying to Dr. Bothman, my experience has been that we did not get as good results from the Gonin as we did from the Lindner operation, as Dr. Suker prefers to call it. I disagree with Dr. Sukar on the comparison between results now and years ago. I think we are getting better results now, with the Guist-Lindner operation. Dr. Suker's point about a stenopeic lenses is important. I think they should be used in every case.

We should operate on these cases early, as the results are much beter. Gonin has recommended operation as early as three or four days. In traumatic cases, I believe we can be more conservative. We should not operate right away, but can afford to wait for a week or two to see if the case will respond to the non-operative treatment satisfactorily.

TRICHOMONAS VAGINALIS

A Report of 300 Cases in Pregnancy With Complete Puerperal Records

EDWARD A. CROWN, M. D.
Former Resident in Obstetrics, Cook County Hospital
CHICAGO

The present study, which includes complete prenatal and postnatal records, was undertaken to determine the role of Trichomonas vaginalis in pregnancy, labor and the puerperium. The material for this investigation was obtained from the prenatal clinic of the Cook County Hospital at Chicago, the patients having been selected at random.

Although Trichomonas vaginalis was described by Donné almost one hundred years ago, it is only in recent years that much consideration has been given to this organism. In the recent literature, we find various opinions as to the incidence and pathogenicity of the organism and its supposed effects. Conflicting statements have been made by many investigators, especially in regards to the relation of Trichomonas vaginalis to pregnancy and the puerperium.

That Trichomonas vaginalis is a common invader of the vaginal tract is now an accepted fact. The role of this organism in the vaginal flora is, however, very little understood.

Trichomonas vaginalis is a pear shaped protozoan, slightly larger than a polymorphonuclear leucocyte, having free flagella at the anterior end, an undulating membrane extending to near the middle of its body, and an axostyle, posteriorly.

In fresh material the protozoa are extremely active, moving with a jerky forward motion and spinning counterclockwise on their long axis. Observing the protozoan on the slide, degeneration occurs, but active motion is often present for several hours. Studying the protozoan after marked degeneration has taken place, we find that the flagellate has assumed an ameboid appearance with pseudopodia extruding from its surface, and its body being shrunken. As degeneration proceeds the motility diminishes. Very often slides made immediately after withdrawal of the discharge show Trichomonas simulating the above pictured organisms with irregular outlines and sluggish movements, or spinning on its long axis without forward motion. Whether these organisms are in a process of degeneration or a mutated form produced by an unfavorable environment, or perhaps are of a different strain remains to be proved, but it is significant that they are generally found in those cases which present no symptoms.

In this series, all examinations were made from fresh hanging drop slides by diluting a small amount of vaginal discharge (which was obtained by speculum) with warm saline solution. Under the high power, the Trichomonas vaginalis were easily seen and were usually plentiful when present. In about 14% of the cases, however, diligent search did not reveal more than one or two organisms in an entire slide, and often only after exhaustive examination of repeated slides.

Accompanying the Trichimonas vaginalis, one

sees in addition to the usual debris and epithelial cells, some leucocytes and a fair amount of bacteria, mostly motile rods. After the examination of many slides, one is impressed with the apparently definite relationship of these three constituents and it is noteworthy that in those cases in which the leucocytes are very few or the bacteria excessively numerous, the protozoan is usually not found.

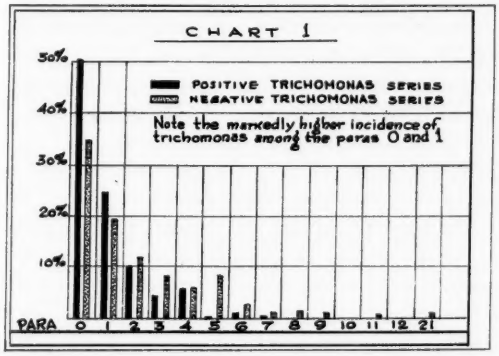


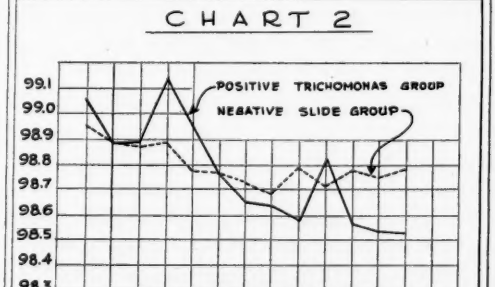
CHART 1
POSITIVE TRICHOMONAS SERIES
NEGATIVE TRICHOMONAS SERIES
Note the markedly higher incidence of trichomonas among the paras 0 and 1
50%
40%
30%
20%
10%
PARA 0 1 2 3 4 5 6 7 8 9 10 11 12 21

Incidence. Statistics based on recent investigations show the incidence of Trichomonas vaginalis to range from 6% to 75%. In this series, it was interesting to note that at times both extremes were reached for rather long periods, and had the series been small, a figure approaching either of the extremes would have resulted.

One of the three hundred cases examined, one hundred were found to be positive for Trichomonas vaginalis giving a general incidence of 33%. 200 of these women were white, and of these 50 or 25% showed positive slides. Of the 100 colored women 50% were positive for Trichomonas. This markedly higher incidence among the colored women is in keeping with reports from other investigators, and is as yet unexplained. It is probably due to a difference in personal hygiene. This same factor probably is responsible for the variations in the reported incidence of Trichomonas vaginalis since the class of patients examined may vary in the different series.

Para and Gravida? At the time of examination, the patients in this series were all in their ninth month of pregnancy. Of the patients with Trichomonas 51% were nulliparas, 24% were primiparas, 10% para three, etc., a positive slide being found in one para thirteen. Of the negative series only 35% were nulliparas, 20% primiparas, etc. A study of table 1 will show that Trichomonas vaginalis was much more common in the nullipara than in the other types. It appears incongruous that a supposedly pathogenic organism should be encountered more frequently in nulliparas than in multiparas. This finding also tends to cast doubts upon the statements of Dubrovin[2] and others that the source of invasion may be through sitz baths or other similar routes; for in that case infection would surely be more common in the women whose genitals were more gaping thereby affording easier entrance to the organism.

Checking to see what role Trichomonas may play in the production of abortions, it was found that in the 51 nulliparas of the positive series, this was their first pregnancy in all cases. Of the 70 patients having negative slides, 10 had had one or more abortions. In the primiparas there was a slightly higher incidence of abortion in the positive Trichomonas group. The significance of this finding cannot be evaluated at this time, but it is certainly unusual to find 51 gravid nulliparas (picked at random) not a single one of which had ever had a previous conception. While this evidence is too meager to be entirely convincing, it does, when coupled with previously stated facts, tend to make one believe that Trichomonas vaginalis may play a very useful role in the vaginal flora.



Age. The ages of the patients in this series ranged from 16 to 48. The average age of the group positive for Trichomonas was 23.3 years as against 26.3 years for those patients whose slides were negative. This apparent difference is in part due to the fact that the positive group had more nulliparas and therefore more patients whose ages we would expect to be lower. On the

whole as has been pointed out before by other investigators, age was not an important factor.

Symptoms. Davis[3] states that he did not find a single case harboring Trichomonas vaginalis which was entirely free from symptoms of leucorrhea or vaginitis. Bland[4] reports that only 13% of the patients complained voluntarily of symptoms but that a larger number admitted symptoms upon questioning.

In this series the vast majority of the cases were without symptoms. 15% of the patients complained of leucorrhea. 4% complained of soreness. Physical findings on three of these patients elicited a sub-acute vaginitis, the fourth patient having a very severe vaginitis. There are the cases which are described as Trichomonas vaginalis vaginitis. This distinct vaginitis was manifest in only 4% of the patients harboring the protozoan. Dysparunia, as may be expected, was a constant symptom in the patients who had a picture of inflammation.

The discharge is usually described as being profuse, thin, yellowish, bubbling or foamy, and having a marked irritating quality which is responsible for chafing and irritation of the external genitals. This was found to be the usual picture in the patients who had a marked discharge. However, as previously stated only 15% of the patients had marked discharge. In the majority of cases the secretion differed from the above picture, the organism being found in almost every type of discharge.

Labor. The conduct of labor was exactly the same for both groups. A record was kept to determine any differences in presentation or in the course of labor.

As far as presentations are concerned there was no difference except that there were six L. O. P. presentations in the normal group and none in the Trichomonas group. This may undoubtedly be ascribed to coincidence. As to the course of labor, the labors of the patients with Trichomonas were of somewhat longer duration, but this difference is only apparent because of the greater number of nulliparas in this group. Checking the labors of the nulliparas in both groups there is no appreciable difference in the duration of any of the three stages of labor.

Obstetrical maneuvers were completed without knowing the patient's status as to Trichomonas and the result was a slightly lower incidence of accouchers' force among the patients with positive slides. The only difference which I am able to note is increased pain in those patients having definite vaginal inflammation. This would naturally be expected. It is interesting to note that the one case having a marked acute vaginitis failed to heal after epesiotomy, and was the only case in which sloughing occurred.

Puerperium. The prime purpose of this study was to determine whether or not Trichomonas vaginalis constituted the grave post partum danger ascribed to it by Bland,[5] Schmid and Kamniker,[6] and other recent investigators.

Both groups of patients received the same puerperal treatment. They were administered to by the same attendants, the patients being housed in adjoining beds of the same ward. The puerperal data was obtained by reviewing the patients' charts after discharge from the hospital. The peak temperature of the patient for each day was recorded and a daily average of these peaks was obtained for both groups. It is from these figures that the curves shown in Chart 2 were made.

An analysis of the graph shows the positive Trichomonas group to have a rise on the fourth day and then rapidly subside, reaching a much lower level than the normal group by the end of the 13th day. From a somewhat more liberal standpoint, the graphs may be said to almost coincide since the greatest variation is less than 0.3 degrees.

In order to have a more detailed picture of the cases with temperatures, I isolated all cases of each group having temperatures of 100° or over at any time. Of these there were 17 in the group of positive Trichomonas and 30 in the negative group giving a percentage of 17 and 15 respectively. Table 1 explains the cause for the temperature in some of the cases.

A review of the causes of these temperatures (where cause could be determined) shows that the Trichomonas was not the causative agent for the slightly higher incidence of these temperatures in its group.

I believe that the evidence given covering as it does a fairly large number of cases, absolves the Trichomonas as a causative organism in puerperal morbidity.

SUMMARY AND CONCLUSIONS

300 patients in their ninth month of preg-

nancy were examined for Trichomonas vaginalis. 50 out of 200 white women and 50 out of 100 colored women were found to harbor the organism.

The incidence of Trichomonas vaginalis was found to be much greater in nulliparas than in multiparas although age is apparently an irrelevant factor.

The large majority of patients with Trichomonas vaginalis had no complaints.

Trichomonas vaginalis may be found in almost any type of discharge, the characteristic discharge being thin, yellowish and foamy.

Trichomonas vaginalis is not a causative agent in abortion.

Trichomonas vaginalis does not cause increased difficulty during labor unless there is an associated vaginitis.

Trichomonas vaginalis does not cause or increase puerperal morbidity.

1150 North State Street.

BIBLIOGRAPHY

1. Donné, M. A.: Animalcules observés dans les matierès purulentes et le produit des sécrétions des organes génitaux de l'homme et de la femme. Compt. rend. Acad. d. sc., 1836, iii, 385-386.

2. Dubrovin, K. M.: The Problem of Pathogenesis of Trichomonas Vaginalis and the Treatment of Colpitis, Omsky Meditzinskiy Journal, 4:28-36, 1929.

3. Davis, Carl Henry: Trichomonas Vaginalis, Amer. Jour. of Obst. and Gyn., 18:575-580 (October), 1929.

4 and 5. Bland, P. Brooke, Goldstein, Leopold and Wenrich, David H.: Vaginal Trichomoniasis in the Pregnant Woman, The Jour. A. M. A., 96:157-163 (January 17), 1931.

6. Schmid, A. L. and Kamniker, H.: Trichomonas Vaginalis, Archiv. für Gynakologie, 127:362-383, 1925-1926.

7. Greenbill, J. P.: The Treatment of Trichomonas Vaginalis Vaginitis, The Jour. A. M. A., 96:1862-1865 (May 30), 1931.

8. Cornell, Edward L., Goodman, L. J. and Matthies, Mabel M.: The Culture, Incidence and Treatment of Trichomonas Vaginalis, Amer. Jour of Obst. and Gyn., 22:360-268 (September), 1931.

9. Sayer, Joseph H.: Vulvovaginitis Pruritic Trichomonalis, Northwestern Medicine, 30:278-289 (June), 1931.

10. Andrews, C. J.: Trichomonas Vaginalis Vaginitis, Virginia Medical Monthly, 58:671-674 (January), 1932.

11. Faulkner, Robert L.: Trichomonas Vaginalis, The Ohio State Med. Jour., 26:765-767 (September 1), 1930.

12. Holmes, Walter R.: Trichomonas Vaginalis Vaginitis, The Jour. of the Med. Assoc. of Georgia, 21:23-26 (January), 1932.

13. Kleegman, Sophia J.: Trichomonas Vaginalis Vaginitis; a Common Cause of Leukorrhoea, Surg. Gyn. and Obst., 51:552-555 (October), 1930.

14. Kamperman, George: Trichomonas Vaginalis Vaginitis, The Journal of the Michigan State Medical Society, 30:686-688 (September), 1931.

15. Evans, John R.: Trichomonas Vaginitis, Colorado Medicine, 29:59-63 (February), 1932.

16. Mathieu, Albert: Trichomonas Vaginalis, Northwest Medicine, 29:15-18 (January), 1930.

TABLE 1

Normal Group	KNOWN COURSE OF TEMP. Analysis of Cases Having Temperature Above 100°	Pos. Trich.
0	Breast Abscess	1
0	Protracted Labor. Forceps unsuccessfully applied	3
		1
0	Protracted Labor. Craniotomy	
1	Protracted Labor. Dystocia	1
0	Stillbirth	0
2	Puerperal Sepsis	0
0	Metritis	1
0	Twins. Added manipulation	1
0	Infected Episiotomy	1
1	Bronchopneumonia	0
2	Pyelitis	0
1	Uterine pack for hemorrhage	0
1	Forceps	0
1	Cesarean Section	0
1	Previous Section. Now delivered from below	0

MEDICAL LEADERSHIP AMONG LAY GROUPS*

LENA K. SADLER, M. D.
CHICAGO

In this paper I wish to present to the Secretaries of the County Medical Societies a plan which is designed to combine the community health activities of lay groups into county units under medical leadership, thus avoiding duplication of effort as well as enabling the various interested agencies of the county to work efficiently along definitely planned lines.

The first step in this program is the full understanding and approval of the County Medical Society, who appoint a committee of five members who are enthusiastic about the plan. One member of the Medical Committee is to serve as chairman of the general county committee.

The membership of this general county committee consists of a representative of organized medicine (the chairman of this small medical committee who has already been appointed), organized dentistry, the Illinois Federation of Women's Clubs, the Illinois Parent-Teacher Association, the county and the larger city superintendents of schools, and, not to exceed three other representatives of active local groups.

This county group should operate under the leadership of the chairman of the medical committee, with the other members of the medical committee serving in an advisory capacity to the chairman. In general, it may be well to choose as secretary of this county health council, a

*Presented before the Secretaries' Conference at Peoria, May 16, 1933.

Federated Club woman because her State Chairman is a medical woman.

It has been found most effective to suggest that each of the representatives have a sub-committee. Thus the County Superintendent of Schools could have a committee on schools, and so on.

Once the group is organized, the next step is to decide on the program. The most practical procedure is for the Advisory Committee from the County Medical Society to invite the State Department of Public Health to help them survey the county and find its actual needs. There may be found need for 1. immunization against diphtheria; 2. vaccination against smallpox; 3. examination of the preschool child; 4. education in prenatal care; 5. restoration of crippled children; 6. educational work along the lines of giving information regarding the prevalence of rabies, eye infections, etc.

It may be the decision of the county health council to work on one project at a time until such work is cleaned up in the county, or it may be found desirable to select several lines of activity. At any rate, a definite program should be worked out, plans of procedure decided upon, certain portions of the work allotted to the various members, and all details thoroughly settled on that there may be concerted action.

Very naturally many matters peculiar to the community will come up and some will probably be difficult to settle, but can be satisfactorily ironed out with frank discussion, a sincere desire to cooperate, and an earnest effort to attain a definite objective.

It is the policy that every parent who is not dependent should pay a fee for services rendered. The plan provides that the Medical Society should immunize, vaccinate, or conduct other repair work—whether it be the removal of tonsils and adenoids, the correction of dental defects, or the management of crippled children, in the manner they decide most suitable to the situation, in their offices, at schools, in clinics.

Each organization represented on this county health council should work to find the children who are handicapped or who need examination or medical attention. Of course, certain particular duties will naturally fall to each of the organizations fitted by their nature for certain phases of work.

With the personnel of the county health council selected, the needs of the county analyzed, the projects chosen, the next step is the planning of publicity to stimulate interest in the activities of the council. Through the Superintendent of Schools various meetings could be arranged, lectures planned through the publicity chairman, speakers sent to the various clubs in the county. The newspapers could be supplied with interesting material regarding the health projects. The Chamber of Commerce, the men's service clubs, and the churches all offer opportunities for emphasizing the value of this work.

The State Health Department and the Educational Committee of the Illinois State Medical Society will assist in providing lectures, moving picture reels, charts, exhibits, etc., and the supervising nurse in the district will give unlimited support to the county council. A medical representative of the Division of Child Hygiene, State Department of Public Health, will be glad to meet with the council, answer questions, and give any advice desired.

THE DIAGNOSIS AND TREATMENT OF ANEMIAS OF INFANCY*†

ARTHUR F. ABT, M. D.
CHICAGO

In the past few years a great deal of interest has been attracted to the general subject of anemic conditions, and more attention is being paid to the anemias of infancy. Before we may adequately discuss this subject, the normal hemoglobin and red cell values for the newborn period and the first months and years of life should be ascertained.

The fetus in utero is an environment of low oxygen tension, which accounts for the polycythemic blood with high hemoglobin and red cell values which the newborn infants exhibit. It is not unusual at birth to find 120 per cent. of hemoglobin and over six million erythrocytes in the circulating blood of the newborn. This polycythemia is a compensatory mechanism on the part of the fetus, to offset the lowered oxygen tension of its intrauterine life, just as an acclimatization to high altitudes and lowered oxygen tension, causes a polycythemic blood to accom-

*From the Department of Pediatrics, Northwestern University Medical School, Chicago.

†Read before the Section on Medicine of the Illinois State Medical Society, Peoria, May 17, 1933.

modate the organism to such an environment.

With birth the infant is delivered into the increased oxygen tension of atmospheric air, and there is a gradual reduction of hemoglobin and red cell values, most rapid in the newborn period. All evidence points to an increased hemolysis of circulating erythrocytes in the newborn period. The liberated hemoglobin is in turn broken down into bilirubin and iron. Simple icterus neonatorum is probably an indication of this excess hemolysis, and an increased indirect Van den Bergh reaction and high ictrus index in the newborn period substantiate this fact.[1]

The iron depot theory of Bunge[2] which assumed that iron was stored in the fetal liver during the last few months of intrauterine life to be used by the infant during the first six to twelve months of its life, as a source of the iron for new hemoglobin formation, is probably incorrect. Bunge's theory was also used to account for the more rapid drop in hemoglobin and erythrocyte values noted in premature infants, the so-called "physiologic anemia of prematures," by assuming that the premature birth interrupted the iron deposition in the fetal liver.

Actually, the livers of newborn full term or premature infants contain little iron.[3] As hemolysis of the polycythemic blood progresses and the liberated hemoglobin is broken down into bilirubin and iron, the liberated iron is in part picked up and stored in the liver, spleen and other organs, and in part is excreted through the intestinal tract.[4] This iron may again be utilized to form new hemoglobin as the need arises; just as in an internal hemorrhage new blood formation progresses rapidly as the products of the extravasation are resorbed and reutilized.

The first step in the drop of the high hemoglobin and red blood cell values present at birth, is the increased hemolysis occurring during the first few weeks of life, due to the acclimatization to the increased extrauterine oxygen tension. As the infant rapidly grows to double its birth weight, the total blood volume must increase correspondingly. Together with and following the early destruction of blood in the young infant, there must follow a rapid formation of new blood to supply the necessary elements for the increasing blood volume corresponding with the infant's rapid growth.

The early hemolysis of red blood cells ceases, and the bone marrow becomes highly active, using as building stones for new blood formation the products of the early destruction of erythrocytes. Thus the excess of iron picked up and deposited in the liver and spleen is again utilized to accommodate for the rapidly increasing blood volume, and sometime after the fourth month of life the excess of iron has been used up. It will then be necessary to obtain sufficient iron from the diet for future blood formation.

The premature infant, born with a smaller total blood volume than the full term infant, and growing at an increased rate, simply exaggerates the normal process in the full term infant. New blood formation may be somewhat less active due to the relative immaturity of the blood forming organs. This is substantiated by the greater degree of anemia in those infants of 6 to 7 months gestation, than in those of 8 months gestation.[5] The more premature the infant the more severe the anemia which will develop. Thus, in the premature we have less total blood to start with; a polycythemic blood at birth, a rapid hemolysis and blood destruction for the first few weeks, a more rapid growth than the full term infant with a greater demand for blood formation to accomodate the increased blood volume, and somewhat immature blood forming organs to fulfill the demands. The total amount of iron and building stones is less than in the full term infant, and probably more rapidly used up somewhere from the eighth to tenth week of life, when the hemoglobin and erythrocyte values are approaching their lowest values.

These normal decreases in the hemoglobin and red cell values can best be graphically shown.

Bearing in mind these normal figures for hemoglobin and red blood cell values, we may now consider some of the anemias of infancy.

A. ANEMIA OF THE NEWBORN

Anemia may manifest itself in the newborn infant, and under the title, erythroblastosis of the newborn two clinical entities have been described, "Anemia of the Newborn, and Icterus Gravis Neonatorum with Erythroblastosis."

It would be only natural to search for a congenital cause for the anemia of a newborn infant, and some illness on the part of the mother might explain the anemic condition of her newborn infant. However, well authenticated cases of blood disease or anemia on the part of the

mother have not been transmitted to the newborn infant. Even pernicious anemia on the part of the mother has not been transmitted to the infant, and authentic cases of leukemia in pregnancy have terminated uneventfully, as far as the infant was concerned.[6] Recent studies have also shown that the anemias of pregnant mothers are not transmitted to their offspring.[7] Anemia of the newborn has been used to designate a group of reported cases in which newborn infants of healthy parents are born in normal labor, and from whose history hemorrhage or loss of blood has been carefully excluded.[8] These babies become profoundly anemic during the first fourteen days of life. Pallor of a sheet-like whiteness may be the only symptom, though reports of jaundice and slight edema have also been noted, as well as a familial history. Recovery has been observed, both without and with treatment in the majority of the cases reported. The blood picture is at first of a mild hyperchromic type with nucleated red blood cells moderately increased.

The associated jaundice, edema and familial history, together with the autopsy reports and blood picture have led me to group these anemias of the newborn as mild manifestations of erythroblastosis fetalis.[9]

B. ERYTHROBLASTOSIS IN ICTERUS GRAVIS NEONATORUM

Under the title icterus gravis neonatorum, a group of newborn infants have been reported, who either at birth or a few hours thereafter, develop a rapidly deepening jaundice, associated with anemia, and a great number of immature erythrocytes in the circulating blood. Associated with the jaundice, anemia and erythroblastic blood picture, are familial history, slight edema, extensive extramedullary hematopoietic foci in the liver and various organs, with increased iron deposition, and occasionally a nuclear icterus in the brain. The blood picture is characterized by a severe hyperchromic anemia, with great numbers of immature red blood cells and occasional young white cell forms. Poikilocytosis, polychromatophilia, with stippling and increase in reticulated red cells are noted.

The characteristic blood findings may be summarized as a severe hyperchromic anemia of hemolytic origin with a marked erythroblatsosis.

The icterus index is markedly increased and a biphasic Van den Bergh reaction is present. From present knowledge it may be assumed that the etiology of this condition can be best ascribed to an *embryonal persistence of hematopoisis of erythrocytes in various organs.*[9]

It should be pointed out that actual sepsis in the newborn and congenital syphilis may cause serve anemia and jaundice in the newborn infant.[9]

C. INFECTIOUS ANEMIAS

The anemias that are caused by and accompany the acute infections, especially the simple respiratory diseases, are probably the most common encountered in young infants. The anemia due to infection is however, not an entity, but merely a symptom and will vary according to the direct or indirect effect the infection may have on the blood forming organs. The infection may cause a diminished production of erythrocytes by a so-called toxic suppression of the bone marrow; or a diminution of erythrocytes by increased hemolysis, which would be termed a hemolytic anemia. There may of course be a combination of both mechanisms in any given case.

The anemia in these mild infections is usually of a slight and secondary nature, and will yield quickly as soon as the infection has been cleared up. However, a succession of colds or upper respiratory infections may produce a more severe anemia, and the anemia in turn aggravates the infection through a lowered resistance on the part of the infant, rendering him more susceptible, and the infection more difficult to clear up. Thus the more infection the more the anemia, and the more the anemia the more susceptible the infant is to infection.

It should be noted in this connection, that as long as the active infection is in progress, it is extremely difficult to permanently effect the anemic condition by any form of therapy. It is only after the baby is afebrile and on the way to recovery from his infection, that therapy will be at all effective (See Table 2)

Case I: The infant at birth was a male premature of 33 weeks gestation and weighed 1,690 gms. The baby was born on July 25, 1929, and discharged from the premature station at the age of 5 weeks in excellent physical condition, weighing 2,085 gms. There had been no difficulty in nursing, no cyanotic spells, nor abnormal symptoms noted during the newborn period.

The infant returned regularly to the outpatient department for examination and dietary instruction. At 3

months, egg-yolk was added to the diet and at 4 months, cereal feeding was started.

At 4½ months of age the infant weighed 5,500 gms., and it was first noticed that he was extremely pale. At this time vegetable soup was added to the diet.

At 6½ months of age the infant was still quite pale and iron and ammonium citrate, gr. 1 daily was administered.

At 8 months of age more vegetables and fruits were added to the diet, and at 9 months the pallor was still marked and the infant *had had a mild attack of rhinopharyngitis,* with low fever and slight diarrhea. Liver and beef juice were added to the diet and the iron and ammonium citrate increased to 1½ gr. daily.

At 10 months of age he was still pale and sun-baths were started. At 1 year of age, it was noted that he was still markedly pale, and the iron ammonium citrate which had been continued for 5½ months was stopped, and sodium cacodylate, gr. 1, bi-weekly injections by hypodermic were continued for 1 month.

At 13 months the infant suffered another mild attack of rhinopharyngitis with diarrhea, and the pallor became more intense. At 14 months of age liver and other meats were added to the diet, and at 15 months, spleen marrow extract, 1 dram, three times daily after meals was started.

At 19 months of age, after several upper respiratory infections, the infant showed a lemon-yellow pallor, the gums, lips, buccal and conjunctival mucous membranes were markedly pale, as were the finger nail beds and the ear lobes. The spleen was firm, enlarged one finger breadth below the costal margin. The appetite was poor and the infant not gaining well. However, he was free from colds at this time.

At 20 months of age the spleen marrow extract was discontinued, and liver extract with iron, E. Lilly's, No. 55 was started. In the accompanying Table 2 will be noted the complete blood findings. In 3 weeks a remarkable improvement was noted, both in the infant's appearance and in the detailed hematologic study.

After a subacute infection of a longer period and greater severity the recovery from the anemia may be prolonged. Such an instance may be illustrated by the records from a child who had recovered from a mastoidectomy complicated by a jugular thrombosis and sepsis. (See Table 3.) The anemia was months in yielding after all infection had subsided. The chronic infections are usually accompanied by a low grade anemia, depending on the severity of the infection and the reaction of the individual.

I have recently observed a 3 year old child with a severe chronic dysentery of 5 months duration, accompanied by a marked secondary anemia.

C. INFECTIOUS-ALIMENTARY ANEMIA

Dietary or nutritional errors, which by lowering resistance may be complicated by infection and thus lead to anemia. An excellent example of this association is the anemia which often accompanies a severe rickets. However, simply healing the rickets will not cure the anemia. By healing the rickets and improving the general nutrition, we are also lessening the incidence to infection, and the anemia will then rapidly yield to treatment.

D. ALIMENTARY ANEMIA

The anemias of purely alimentary or nutritional origin have been long considered in etiologic discussions, as having an important place in the classification of the anemias of infancy.

We may recall that following the early hemolysis of excess red blood cells in the newborn period, a certain supply of available iron is present to meet the demands of rapidly increasing blood volume which corresponds to the general rapid growth of the infant in the first half year. We then realize that the building stones for new blood formation may be used up if new materials are not furnished in the diet. Two facts should be here noted. Mackay[10] has observed that those infants with lower birth weights tend to have lower hemoglobin values as they develop. In other words, the baby with the lower birth weight is more rapidly growing, and therefore, will exhibit a greater demand for new blood formation than the heavier, slower growing group.

Further, milk, both human and cow's, is low in iron content (see Table 4). As cow's milk contains less iron, it follows that artificially fed infants tended to show lower hemoglobin curves than wholly breast fed infants. Krasnogorski,[11] in metabolism experiments showed that the iron retention of infants on human milk was greater than that of infants on cow's or goat's milk. It has been generally conceded that goat's milk anemia corresponds to the same essential etiologic factors as cow's milk anemia.[12]

There is good evidence to believe that alimentary or nutritional anemia is caused by a simple insufficiency or lack of blood building material, especially iron, in the diet. The older theories proposed such as hereditary predisposition; injurious products in cow's milk (fat injury of Czerny); lack of vitamines; lack of fresh air and sunlight; and functional weakness of blood forming organs, need no longer be considered.

Just as other imbalances in infant nutrition may produce disturbances, it can be said that

alimentary anemia is not directly due to an injurious effect of cow's milk or any of its constituents. The same anemia may develop in an exclusively breast fed infant.

The whole nature of the process depends on the proper supply of the necessary blood building materials, which the infant on an exclusive long continued milk diet lacks. The error is not one of commission, but rather one of omission. It is not what we are giving the infant, but what we are failing to give him that is causing the anemia.

The early addition to and balancing of infant diets, which is becoming more and more of common usage and knowledge, is greatly reducing the incidence of pure alimentary anemia.

E. VON JAKSCH ANEMIA AND ERYTHROBLASTIC ANEMIA OF COOLEY

The anemia described by Von Jaksch as "Anemia Infantum Pseudo-Leukemia," occurs in infants from six months to two years of age.[13] It is characterized by a severe anemia, leukocytosis, and with many nucleated red blood cells and other immature cells in the circulating blood. An enlargement of the liver and spleen is also present. The condition has been associated with rickets and infection, and is probably a severe form of secondary anemia due to the association of these two conditions.

Erythroblastic anemia is a rare form of anemia first described and named by Cooley,[14] who differentiated it from Von Jaksch's anemia. It occurs in infants of families originating in Mediterranean countries and is of congenital, familial and racial incidence. It is a slowly progressing anemia with large numbers of nucleated red cells in the peripheral blood. There is enlargement of the liver and spleen, a mongoloid facies, and a characteristic change in the bones on x-ray, which show a thinning of the cortices and a widening of the medullary portion, with prominent trabeculation and radiating spicules from the inner table of the skull.

Anemias with Leucopenia. In this group, there are reported four separate types of cases; agranulocytic anemia with angina; aplastic anemia; aleukemic leukemia and sepsis with leucopenia. All of these types begin with an insidious onset, and later develop fever, pallor and progressive anemia with leucopenia. The course is generally fatal within several weeks to months. Differentiation may be extremely difficult, as has been pointed out by Bigler and Brennemann.[15] In cases of aleukemia Isaac A. Abt[16] has noted the extreme difficulty of differentiation from aplastic anemia, which may be possible only at autopsy.

Treatment of Anemia. In all cases of suspected anemia in infants, it is essential, as has been pointed out, to consider the normal decline of hemoglobin and erythrocytes for the various age periods. Further, the less the birth weight and the more rapid the growth, the greater the expected lowering of these values may be. Breast fed infants on the whole, may have anticipated higher values than artificially fed babies.

Whether the mechanism be infection with blood destruction and toxic suppression of the bone marrow, or omission of blood building materials from the diet in nutritional anemia, the general form of treatment will follow the same outline.

As has been shown in anemia due to infections, all attempts at treatment are useless until the acute infectious process has been overcome.

The use of iron in the treatment of anemia is of ancient origin, and recently there has been a tendency toward the use of larger doses than formerly. Iron may be given as reduced iron, ferrous and ammonium citrate or saccharated ferrous carbonate in doses of from 0.5 gm. to 1 gm. daily. Organic iron compounds are no more effective than the inorganic. Copper has no effect on red cell formation and in the absence of iron it is valueless in the treatment of anemia. In combination with iron it is said to cause a rise in hemoglobin. It may be given as a ½ per cent. solution of crystalline copper sulphate, and in the proportion of 1 cc. per kilogram of body weight. Others recommend 20 mgm. of copper per dose, combined with iron.

Liver and liver abstract have been used in pediatrics for the treatment of anemia since 1912, when Czerny and later Kleinschmidt recommended the usage of liver soups as a treatment for alimentary anemia and as a substitute for milk protein in cases of exudative diathesis. A decade later, Minot first proposed the use of liver for the treatment of primary anemias and Whipple developed the secondary anemia fraction of liver extract.

There are many preparations of liver, iron and copper now available for the treatment and prevention of infantile anemias.

Liver extracts; liver extract and iron or copper; desiccated hog stomach with iron; iron and copper; malt and iron extracts; cereals containing alfalfa and bone meal to increase iron content; spleen marrow extracts; and even a sugar to which iron and copper have been added are all available, and every clinician has his favorite preparation. I know of one clinician of long experience, who favors cooking oysters in the baby's soup on account of the prodigious liver of this mollusk, as his favorite method for preventing and curing infantile anemia.

Transfusion, intravenous or intraperitoneal, is indicated in the severe secondary anemias after hemorrhage; in grave blood diseases where there is a failure of the blood making mechanism to produce sufficient new cells. Also in those conditions where there is active hemolysis transfusion is indicated, and in the infectious diseases associated with anemia where one hopes to combat the infection as well as the anemia, to introduce new antibodies and to tide over a critical period. Some have found intramuscular blood injections of value and others recommend intraperitoneal injections of iron.

TABLE 1. CLINICAL CLASSIFICATION OF ANEMIAS OF INFANTS

1. Premature anemia—twins—full term
2. Erythroblastosis—anemia of newborn
   (a) upper { Rhinopharyngitis; Otitis
3. Infectious anemia: A. Acute: 1. Respiratory
   (b) Pneumonia { Grippe; Bronchitis
   2. Pyelitis, nephritis, furunculosis, dysentery, typhoid, etc.
   3. Sepsis
   B. Chronic:
   1. Tuberculosis
   2. Congenital syphilis—early, severe late, mild
   3. Chronic pyogenic infections, etc.
4. Infectious—Alimentary Anemia
   Nutritional errors plus infection
5. Alimentary:
   1. Cows' milk anemia
   2. Goats' milk anemia
   3. Unbalanced Iron poor diet
6. Von Jaksch—Anemia—Erythroblastic anemia (Cooley)
7. Specific Diseases
   1. Toxic—lead, pot, chlorate, benzol and other poisons
   2. Parasitic—Tape worm—hook worm—malaria
   3. Neoplasms
   4. Splenomeagalies
   5. Scurvy—purpura
   6. Sickle cell
8. Hemorrhage—acute blood loss

TABLE 2. ANEMIA WITH RAPID CURE AFTER RESPIRATORY INFECTIONS

Age & Date	Birth 7-28-29 3-10-31	4-9-31	4-30-31	5-21-31	6-23-31
R. B. C.	3.6	3.4	5.0	5.2	5.3
HGB.	36%	36%	85%	84%	80% I (Tallquist)
W. B. C.	12,300	15,000	14,500	12,900	.....
Plat.	200,000	240,000	.....	.....	340,000
P. M. N.	35%	32%	37%	46%	27%
Lymphs.	57%	63%	56%	43%	65%
Eos.	1%	0	1%	2%	1%
L. Mono.	7%	5%	6%	9%	7%
Bas.	0	0	0	0	0
Nucl. Red.	0	0	0	0	0
Retic. in %	.25%	1%	.25%	0	0
	Birth Wt. 1690				
Infants Weight	9240 grams	9415	10,250 Gms.	10,250	10,500 grms.
Gestation	33 wks. Premature				
Race	White				
Remarks	Marked Pallor 20 months of age	Liver & Iron medication begun	Marked improvement in infants color, etc.		Liver Extract & Iron 1 dram (4 grams) contains Liver Extract 3.75 grams Ferrous Amm. Cit. 0.65 grams (10 grains)

TABLE 3. ANEMIA WITH SLOW RECOVERY AFTER MASTOIDECTOMY AND JUGULAR THROMBOSIS

Date	4-13-32	4-25-32	5-16-32	5-27-32	6-6-32	6-13-32	6-27-32
Medication	Liver, Iron and Copper Sahli	Liver, Iron and Copper Sahli	Liver, Iron and Copper Sahli	1 dram (4 grams) contains Copper Sulphate 0.20 mgm.		Liver Extract 3.49 grams Ferric Amm. Cit. 0.51 grams (8 gr)	
Hemoglobin	53 Units	56 Units	52 Units	57 Newcomer	66 Newcomer	65 Newcomer	65 Newcomer
Erythrocytes	2,380,000	3,100,000	3,500,000	3,340,000	3,800,000	3,780,000	3,940,000
Leucocytes	6,100	12,100	12,000	9,000	9,500	6,200	6,800
% Neut. Poly.	66	68	66	53	60	52	57
% Eos. Poly.			3	2	2	7	4
% Basophiles						2	
% Lymphocytes	34	30	30	41	30	32	35
% Monocytes		2	1	4	8	7	4
% Reticulocytes				5	.25	.25	
Stippled Reds				1			
Remarks				Child had cold			

TABLE 4. VARIOUS FOODSTUFFS SHOWING IRON CONTENT M G M % OF IRON

1. Milk, Human	0.020	Egg Yolk	4.5 to 8.0
2. Milk, Goats	0.016	Beef Liver (fresh)	8.3
3. Milk, Cows	0.014	Beef Liver (dry)	29.4
4. Spinach	3.0 to 4.0	Hog Liver (fresh)	25.0
5. Lettuce	0.66	Hog Liver (dry)	80.0
6. Peas	1.49	Oysters (fresh)	7.6
7. Carrots	0.20	Oysters (dry)	76.0
8. Almonds	3.2	Strawberry	0.65
9. Rye Bread	1.9	Orange Juice	0.02
10. Oatmeal	3.8	Tomato	0.32

BIBLIOGRAPHY

1. Anselmino, K. J. and Hoffman, F.: München Med. Wchnschr., 79: 1226 (July 29), 1932.
2. Bunge, G.: Lehrbuch der Physiol. u. Pathol. Chemie, ed. 2, Leipsig, Vogel 1889.
3. Gladstone, S. A.: Am. J. Dis. Child., 44:81, July, 1932.
4. Lichtenstein, A.: Acta Paediat., 1:194, 1921.
5. Abt, Arthur F. and Nagel, Beth R.: Prophylaxis of the Anemia of Premature Infants, J.A.M.A., 98:2270, June 25, 1932.
6. Baar, H. and Stransky, E.: Klinische Hematologie, Leipsig, F. Deuticke, 1928.
7. Strauss, M. B. and Castle, W. B., Anemia in Pregnancy, Am. J. Med. Sci., 185:539, April, 1933.
8. Abt, Arthur F.: Anemia of the Newborn, Am. J. Dis. Child., 43:337, February, 1932.
9. Abt, Arthur F.: Erythroblastosis in Icterus Gravis Neonatorium, J. Pediat., 3:7, July, 1933.
10. MacKay and Goodfellow: Report No. 157, Medical Research Council, London, 1931.
11. Krasnogorsky, N.: Jahrb. f. Kinderhl., 64:651, 1906.
12. Finkelstein, H., Jahresk. f. Ärtzl. Fortbild., 23:1, June, 1932.
13. Abt's Pediatrics, Chapter on The Anemias, Vol. IV, 583, Saunders, Philad., 1924.
14. Cooley, T. B.: (a) Am. J. Dis. Child., 36:1257, December, 1928. (b) Ibid. J. Paediat., 1: 635 to 651, November, 1932.
15. Bigler, J. A. and Brenneman, J.: Sepsis with Leukopenia, Am. J. Dis. Child., September, 1930.
16. Abt, Isaac A.: Aleukemic Leukemia, Med. Clinics North America, Saunders, Philad., Vol. 8:427, September, 1924.

INTRAVENOUS UROGRAPHY

In the diagnosis of tuberculosis of the kidney by intravenous urography, Pask carries out the injection as in any intravenous injection, using an ordinary 20 cc. syringe and injecting the contents slowly. It has been his practice to take a series of roentgenograms at intervals of five, ten, twenty and forty minutes after the injection, because in a single roentgenogram some part of the ureter or pelvis, or both, will be in systole, and that part will not contain any of the contrast substance. The only parts delineated in a given roentgenogram are those that are in diastole at the time of taking the urogram. The dynamics are constantly changing, owing to the peristalsis. By intravenous urography, visualization of both sides of the urinary tract and the relations between the various parts can be studied in a series of roentgenograms. It is thus possible to demonstrate the presence of both kidneys and to contrast their relative functional activity. It has been proved by animal experiment that neo-iopax (known abroad as uroselectan B) is excreted by the glomeruli of the kidneys and not by the tubules and that before it passes into the renal pelvis, the general outline of the kidney is often well seen because the contrast substance is present in the glomeruli of the kidney. As the neo-iopax passes to the calices and pelvis and along the ureters, any abnormality can be noted. In cases of tuberculosis of the kidney, there is frequently seen irregularity of the outline of the pelvis and calices and dilatation of the ureter. If the kidney substance is completely destroyed on one side, no urogram will be obtained on that side. Neo-iopax, 5 per cent, in the urine, is said to be sufficient to give a roentgenogram. In normal kidneys a good roentgenogram is sometimes obtained as early as five minutes after injection, but in the tuberculous kidney the rate of excretion is retarded and occasionally six hours is necessary before a sufficiently dense pyelogram is obtained.

TREATMENT OF COLITIS

To establish sound treatment of ulcerative colitis, Smith emphasizes that the diagnosis should be based on a careful history and examination of the stools, a sigmoidscopic examination performed without an anesthetic and with a minimum of inflation, and an ex-

amination of a swab taken directly from the ulcerated or inflamed surface of the colon. During the past three years the author has treated six cases of ulcerative colitis with Bargen's serum. The patients havve all been women, and the disease had been present for periods varying from six months to three years. Two were treated by intramuscular injections and four by the intravenous route. The amount of serum used varied from 15 to 50 cc. in divided doses. In all six patients the loss of weight up to the commencement of treatment was 35 pounds (16 Kg.) or more, and all have regained their lost weight except one, who has regained 21 pounds (9.5 Kg.) and is still gaining. In the intravenous method of administration the anaphylactic shock is sometimes alarming. The intravenous injection of from 1 to 2 cc. of 1:1,000 solution of epinephrine immediately after the serum has been given is the best method of minimizing the shock. The shock is not very distressing until 15 cc. of serum is injected. When the reaction is severe, it takes about an hour before the patient feels comfortable. With the intramuscular route there is practically no reaction. After preliminary preparation of the patient, serum treatment is commenced with 1 cc. of Bargen's ulcerative colitis antistreptococcus serum. The dose is increased gradually every day or every second day until 5 cc. is reached. As improvement progresses, the time interval between the injections can be increased, although not more than three days should be allowed to elapse, as the shock is more profound after a longer interval. The shortest period of time required for clinical cure has been nine days, and the longest thirty-six days. This does not represent the length of time required to bring the patient to a state of physical fitness but only the length of time necessary to check the diarrhea. The author has seen these patients regularly over periods ranging from six months to three years, and so far no relapses have occurred.

CHRONIC SUPRARENAL INSUFFICIENCY

Packard and Wechsler report a case of malnutritional edema which exhibited an unusual clinical syndrome and a degenerative lesion of the suprarenals at necropsy. The puzzling feature of the case is the fact that, in spite of the continuation of his diet and for no reason that could be demonstrated by clinical and laboratory examinations, the patient, in the second half of chronic suprarental insufficiency in animals prosively. The main features were the complete loss of body fat and an advanced degenerative lesion of both suprarenals with necrosis, regeneration, hemorrhages and capillary and venous thromboses. The author points out the similarity between the syndrome and the state of chronic suprarental insufficiency in animals produced by bilateral suprarenalectomy. They review the literature describing the effect of total and partial inanition and of the various vitamin deficiencies on the suprarenals in both animals and man and suggest that this clinical syndrome, heretofore undescribed, is one of chronic suprarenal insufficiency due to the suprarenal degeneration occasioned by malnutrition.

WHATEVER ODDS THERE ARE

Give me but room to fight my way,
I ask no other gift from Fate;
Though it should crowd on me at bay,
Where only ghosts and shadows wait.

Shadows of old defeats blown by,
Ghosts of old dreams drawn from life's pit;
Yet all I ask is room to try
And prove Fate cannot make me quit.

No glint of glory from the height,
No flare of fame to call me far;
Merely the ground to make my fight
Against whatever odds there are.

—*Grantland Rice.*

Society Proceedings

COOK COUNTY
CHICAGO MEDICAL SOCIETY

Regular Meeting, November 8, 1933.

CANCER OF THE CERVIX OF THE UTERUS

Laboratory Data, R. H. Jaffé, Pathologist, Cook County Hospital.

Indications for Radiation Therapy, Henry Schmitz, Prof. Gynecology, Loyola University School of Medicine.

The Prevention and Early Diagnosis—Special Features in Treatment (lanternslide demonstration) Arthur H. Curtis.

Sponsored by Cancer Research Institute, Chicago Woman's Club.

Discussion opened by N. Sproat Heaney, Prof. Obstetrics and Gynecology, Rush Medical College.

Regular Meeting, November 15, 1933.

FEDERAL MEDICAL RELIEF FOR THE INDIGENT.

The Federal Emergency Relief Act, William Woodward, Director, Bureau of Legal Medicine and Legislation, American Medical Association.

Local Administration, Roscoe Leland, Director, Bureau of Medical Economics, American Medical Association.

General Discussion, Philip H. Kreuscher, Charles H. Phifer and John R. Neal.

Regular Meeting, November 22, 1933.

ENCEPHALITIS—ST. LOUIS EPIDEMIC.

General Survey, Surgeon Gen. Hugh Cummings, U. S. P. H. Service.

Clinical Data, T. C. Hempelmann, Assoc. Prof. Clin. Ped., Washington University Medical School.

Bacteriological Observations, N. Paul Hudson, Prof. Bacteriology, University of Chicago.

General Discussion opened by Peter Bassoe, Prof. of Neurology, Rush Medical College.

KANKAKEE COUNTY

A regular meeting of the Kankakee County Medical Society was held at Kankakee, Illinois, on November 22, 1933. The program was furnished by members of the State Department of Public Health conducted by the Director, Dr. Frank J. Jirka, who gave a very interesting talk on the following subjects, Epidemic Encephalitis, Tularemia and Amebic Dysentery.

Dr. R. H. Woodruff talked on "Mortality Trends and Vital Statistics."

Dr. H. J. Shaughnessy, "The Doctor and the Diagnostic Laboratory."

There was a large attendance present and the program was very well received; also a closer acquaintance was formed between the members of the State Department of Public Health and the members of the County Society. Dr. W. C. Van Wormer of the State Department was also present.

R. V. Thomas, M. D.,
Secretary.

OGLE COUNTY

Ogle County Medical Society held its Fall meeting at Rochelle, Illinois on Nov. 2, 1933. A fine dinner at Spring Lake was followed by a short business meetting with election of officers.

President, Dr. R. O. Brown, Mt. Morris; Vice-president, Dr. F. G. Andreen, Rochelle; Secretary, Dr. A. R. Bogue, Rochelle; Censor, Dr. G. S. Hendersen, Holcomb; Delegate for 2 Years, Dr. W. E. Kittler, Rochelle; Alternate Delegate, Dr. A. R. Bogue, Rochelle.

Dr. Charles D. Center, president-elect, Illinois State Medical Society, gave a splendid paper on "The Need of Cohesion."

The Disciples of Aesculapius and students of early medicine had a lofty ideal which is being slowly aspirated from the new medical man. The younger men are more and more being workers for pharmaceutical houses and the suave salesman is doing the prescribing. Organization and cohesion are the safeguards to the Medical profession, and the young men should throw off some of their self-satisfaction and look out for the multitudinous encroachments.

Dr. T. B. Knox, Quincy, Councillor 6th District, after a friendly and personal eulogy of the preceding man and a few good stories gave a forceful treatise on cooperation and the effect of the cheap chiseler.

Thirty members and visitors attended and a lively discussion was enjoyed.

A. R. Bogue, Secretary.

Marriages

Robert H. Bell, Carlinville, Ill., to Miss Lydia Reichmann of St. Louis at Indianapolis, September 5.

Hyman J. Burstein, Decatur, Ill., to Miss Edythe Mae Cohen of Springfield, August 27.

Raymond E. Holben, Lincoln, Ill., to Miss Ella De Frates of Springfield, August 28.

Ray Woizeske King to Miss Mary Rose Sherburne, both of Peoria, Ill., August 24.

Robert Edward Lee Gunning, Galesburg, Ill., to Miss Jean Zearing of Princeton, October 7.

Robert Elmer Johnson, Danville, Ill., to Miss Virginia Blunk at Indianapolis, August 31.

Personals

Dr. Max Thorek addressed the McHenry County Medical Society, November 16, on "Non-surgical Treatment of Osteomyelitis."

Dr. Solomon Strouse, Chicago, discussed obesity before the Will-Grundy County Medical Society, November 8.

Dr. Elwood W. Mason has been named instructor in medicine in the division of biological sciences, University of Chicago.

Dr. William E. Buxton, West Salem, observed his seventy-fifth birthday, recently. Dr. Buxton has been practicing medicine for fifty years.

Dr. and Mrs. Charles D. Gardiner, Grand Tower, celebrated their golden wedding anniversary, October 11.

At a meeting of the McDonagh Society for Clinical Research, November 17, among others, Drs. Ernst Pribram and Stanley Fahlstrom spoke on "Studies in Arthritis and Arthrosis."

At a meeting of the Chicago Pathological Society, November 13, the speakers included Dr. Victor Levine on "Myocardial Changes in Essential Hypertension."

At a meeting of the Chicago Pediatric Society, Dr. Abraham B. Schwartz, Milwaukee, among others, discussed "Home Versus Hospital Care of Sick Children."

Speakers before the Chicago Surgical Society, November 3, included Dr. Fred W. Rankin, Lexington, Ky., on "Cancer of the Colon: Notes on Its Surgical Treatment."

The French Government has notified Disraeli Kobak that the Minister of Public Instruction and Fine Arts has signed a decree on October 2, 1933, conferring upon Dr. Kobak the University Palms, Grade Officier d'Academie, in recognition of his contributions to Physical Therapy.

At a meeting of the Bureau County Medical Society in Spring Valley, November 7, Dr. Charles Morgan McKenna, Chicago, spoke on

"Diagnosis and Treatment of Diseases of the Kidney."

Dr. Nathan S. Davis III, Chicago, discussed "Hypertension—Coronary Disease" before the Peoria City Medical Society, November 7.

Dr. Frank Garm Norbury, Jacksonville, discussed psychoneuroses not requiring institutional care before the Adams County Medical Society, November 13, in Quincy.

At a meeting of the Iowa and Illinois Central District Medical Association in Moline, October 26, Dr. Bert I. Beverly, Chicago, spoke on "Behavior Disturbances in Childhood."

Dr. John J. McShane, Springfield, spoke before the Christian County Medical Society in Taylorville, October 25, on epidemic encephalitis and other contagious diseases of the nervous system.

Dr. Ernst Kraas, University of Frankfort, Germany, spoke before the Physiology Journal Club, Northwestern University Medical School, November 16, on "Choice of Operation in the Surgery of the Stomach and Gallbladder."

Dr. Arturo Castiglioni, professor of the history of medicine, University of Padua, lectured in the Italian Pavilion, Century of Progress, November 6; his subject was "The Renaissance of Medicine in Italy."

Dr. Benjamin Goldberg, associate professor in medicine, University of Illinois College of Medicine, was given an honorary professorship in the National University of Mexico, October 30, and gave a series of lectures on tuberculosis in Mexico City.

The Chicago Pediatric Society was addressed, November 21, by Drs. George Piness, Los Angeles, on "Food Factors in Allergy of Childhood," and Louis W. Sauer, "Whooping Cough Immunization."

The Chicago Council of Medical Women was addressed, November 3, by Drs. Minnie S. O. Perlstein and Esther T. Frankel on "Lymphogranuloma Inguinale" and "Use of Ultraviolet Light in Neuralgic Pain," respectively.

Dr. Isaac A. Abt will address the eighteenth annual meeting of the Institute of Medicine of Chicago, December 5, on "Treatment of Whooping Cough: A Study in the Evolution of Therapeutics."

The theme of the meeting of the Chicago Academy of Criminology, November 9, was classification of prisoners and individualization in management; the speakers were Dr. Paul L. Schroeder, Andrew Brown, Ph.D., and Mr. Donald Clemmer.

The Society of Medical History of Chicago was address, November 23, by Drs. James B. Herrick on "Allan Burns: Anatomist, Surgeon, Cardiologist, 1781-1813," and by Hugh T. Patrick on "Three Great Neurologists I Have Known."

Dr. William G. Rogers, among others, addressed the Chicago Gynecological Society, November 17, on "Rupture of the Fetal Liver," and Drs. Beatrice E. Tucker and Harry B. W. Benaron on "Parasacral Anesthesia in Obstetrics." Dr. Fred L. Adair was elected president.

Speakers before the Chicago Neurological Society, November 16, included Drs. Paul C. Bucy on "Ipsilateral Representation in the Motor and Premotor Cortex" and Mabel G. Masten, Madison, Wis., "Neurogenic Ulcers in Cerebral Lesions (Cushing's Syndrome)."

Speakers before the Ogle County Medical Society at Rochelle, October 26, included Drs. Charles D. Center, president-elect of the Illinois State Medical Society, and Thomas B. Knox, sixth district councilor, both of Quincy, on "The Need of Cohesion" and "Medical Care of the Unemployed," respectively.

Speakers before the Southern Illinois Medical Association, November 2-3, at Centralia, included Drs. Llewellyn Sale and Vilray P. Blair, St. Louis, on blood dyscrasias and correction of face injuries, respectively; Walter C. Alvarez, Rochester, Minn., common causes of nervous indigestion, and Harry S. Crossen, St. Louis, inflammatory conditions of the female pelvis.

Dr. William Snow Miller, emeritus professor of anatomy, University of Wisconsin School of Medicine, Madison, opened a series of lectures at the Veterans Hospital, Hines, October 30, with a talk on "The Air Spaces and Their Structure." Other subjects discussed on subsequent days were "The Vascular Supply of the Lung"; "The Lymphatics and Lymphoid Tissue," and "Some Applications of the Above Subjects to Disease."

News Notes

—The health division of the Council of Social Agencies was addressed at its annual meeting,

November 9, by Drs. Frank J. Jirka, state health officer; Frederick Tice of the Municipal Tuberculosis Sanitarium, and Frederick O. Tonney of the Chicago Board of Health.

—The National Research Council, Washington, D. C., has given $21,000 to the University of Chicago for research in problems of sex. The amount will be divided into $8,000 for the investigation of the biochemistry of sex hormones, under the direction of Fred C. Koch, Ph.D., and $13,000 for investigations in the biology of sex under the direction of Dean Frank R. Lillie, Ph.D.

—Dr. Henry Close Hesseltine, instructor in obstetrics and gynecology, Division of Biological Sciences, University of Chicago, was awarded the annual prize of $100 by the Central Association of Obstetricians and Gynecologists for the most meritorious work done by one of its members. This is the second time Dr. Hesseltine has won the prize, this year for his research on "Trichomonas Vaginalis Vaginitis," and last year for a paper on "Gynecologic Fungi Infections in Diabetic Patients."

—The tumor clinic of Michael Reese Hospital is conducting a clinical investigation on massive radium therapy of a special group of neoplasms and will accept free of charge a limited group of indigent patients suffering from these conditions. The tumors under investigation are cancer of the tonsil, pharynx, larynx and prostate, also benign prostatic hypertrophy occurring in patients in whom operation is contra-indicated. It is essential that these patients should not have received any previous radiation treatment. It is requested that the referring physician submit data concerning a case before sending the patient.

Deaths

SAMUEL ALPERT, Chicago; University of Toronto Faculty of Medicine, Toronto, Ont., Canada, 1933; aged 26; intern at St. Joseph's Hospital, where he died, November 2, of brain tumor.

JOSEPH C. BROOKHART, Oak Forest, Ill.; St. Louis College of Physicians and Surgeons, 1903; member of the Illinois State Medical Society; on the staff of the Cook County Tuberculosis Hospital; aged 53; died suddenly, October 17, of heart disease.

JOHN ROBINSON BUCHAN, Chicago; Long Island College Hospital, Brooklyn, 1872; Civil War veteran; aged 87; died, October 27, of toxemia and urinary retention due to carcinoma of the prostate and bladder, and hypostatic pneumonia.

THOMAS FAITH, Chicago; College of Physicians and Surgeons of Chicago, 1893; member of the Illinois State Medical Society and the American Academy of Ophthalmology and Oto-Laryngology; on the staff of the South Shore Hospital; aged 61; died, October 16, of arterioschlerosis and cerebral hemorrhage.

WILLIAM FULLER, Chicago; Rush Medical College, Chicago, 1887; member of the Illinois State Medical Association; formerly professor of operative surgery and associate professor of clinical surgery at the University of Illinois College of Medicine; Fellow of the American College of Surgeons; served during the World War; on the staffs of the Washington Park, Woodlawn and Englewood hospitals; aged 69; died, October 25, of uremia and carcinoma of the prostate.

SARAH LEE GOODWIN, Chicago; Hahnemann Medical College and Hospital, Chicago, 1893; aged 77; died, October 21, in the Ravenswood Hospital, of cardiac decomposition, following a fracture of the femur as the result of a fall.

OSCAR PORTER HARRIS, Mendota, Ill.; Rush Medical College, Chicago, 1902; a Fellow, A. M. A.; physician and owner of a hospital bearing his name; aged 62; died suddenly, October 29, of cerebral hemorrhage.

LYMAN G. HEMENWAY, Sycamore, Ill.; Bennett Medical College, Chicago, 1877; member of the Illinois State Medical Society; aged 80; died, October 5.

JOHN CUNNINGHAM MAXWELL, Sterling, Ill.; College of Physicians and Surgeons of Chicago, School of Medicine, of the University of Illinois, 1901; aged 67; died suddenly, October 14, in Houston, Texas, of cerebral hemorrhage.

JAMES EDWARD MCCARTHY, Hubbard Woods, Ill.; Rush Medical College, Chicago, 1927; formerly on the staffs of the Highland Park (Ill.) Hospital and St. Francis Hospital, Evanston; aged 34; died, October 26, of Hodgkin's disease.

JOHN PAGE MCMAHAN, Peoria, Ill.; Rush Medical College, Chicago, 1883; member of the Illinois State Medical Society; on the staff of the Proctor Hospital; aged 75; died, October 5.

GEORGE HUTCHINS MCNEMER, Cairo, Ill., University of Louisville (Ky.) School of Medicine, 1889; a Fellow, A. M. A.; past president of the Alexander County Medical Society; formerly on the staff of St. Mary's Hospital; aged 67; died suddenly, October 18.

WILLIAM K. POWIS, Chicago; Chicago Medical School, 1928; member of the Illinois State Medical Society; aged 42; on the staff of the Burnside Hospital, where he died, October 8, of acute intestinal obstruction.

JAMES WALTER RENDLEMAN, East St. Louis, Ill.; Jefferson Medical College of Philadelphia, 1894; a Fellow, A. M. A.; past president of St. Clair County Medical Society; on the staff of St. Mary's Hospital; aged 65; died, October 13, of heart disease.

, 1933

ention
hypo-

s and
linois
ny of
aff of
er 16,

llege,
edical
rgery
Uni-
f the
the
Park,
died,
ate.

edical
died,
rdiac
ur as

edical
sician
l 62;
.

nnett
linois

llege
ol of
l 67;
cere-

Ill.;
n the
d St.
tober

edical
State
pital;

ersity
llow,
ounty
Mary's

edical
edical
pital,
truc-

; Jef-
llow,
edical
l 65;